Prof. Dr. Cezmi A. Akdis

Swiss Institute of Allergy and Asthma Research

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Index

About us

Prof. Dr. Cezmi A. Akdis

The Swiss Institute of Allergy and Asthma Research (SIAF) is a department of the foundation Swiss Research Institutes for High Altitude Climate and Medicine Davos (SFI) and an affiliated institute of the University of Zurich and member of the Life Science Zurich Graduate School. The institute in its current form arose from the medical department of SFI in 1988. Since this time the research activities at SIAF are focused on basic research in the field of allergies and asthma.

1905	Tuberculosis Research Institute Davos
	Medical Society Davos, Community of Davos, K. Turban
1907	Physical-Meteorological Observatory Davos, C. Dorno
1922	Swiss Research Institute for High Altitude Climate and Tuberculosis
1922-1933	A. Loewy, High Altitude Physiology
1934-1937	F. Roulet, Chemistry of Mycobacterium Tuberculosis
1938-1954	W. Berblinger, Pathology of Tuberculosis
1954-1960	W. A. Vischer, Resistance to Mycobacterium Tuberculosis
1961	Swiss Research Institute for High Altitude Climate and Medicine
1961-1985	E. Sorkin, Neuroendocrine-Immune Interactions
1985-1987	H. Basedowsky, Neuroendocrine-Immune Interactions
1988	Swiss Insitute of Allergy and Asthma Research (SIAF)
1988-2006	K. Blaser, Mechanisms of Allergy and Asthma
2006-present	C. A. Akdis, Mechanisms and Novel Methods for the Diagnosis and Treatment of Allergy and Asthma

SIAF Wandertag 2025

Bericht des Direktors

Prof. Dr. Cezmi A. Akdis

Das Schweizerische Institut für Allergie- und Asthmaforschung (SIAF) wurde 1988 von der Medizinischen Abteilung des Schweizerischen Forschungsinstituts für Hochgebirgsklima und Medizin Davos (SFI) gegründet. Ziel des Instituts ist es, durch seine Forschung das Verständnis von Allergien und Asthma zu verbessern. Seit 1996 ist das SIAF der Universität Zürich angegliedert und seit 2008 Mitglied der Life Science Zurich Graduate School, einem gemeinsamen Ausbildungsprojekt der Universität Zürich und der ETH Zürich. Diese Anbindung ermöglicht dem SIAF die Durchführung einer umfassenden PhD-Ausbildung. Darüber hinaus ist das SIAF aktives Mitglied der Academia Raetica sowie der Graduate School des Kantons Graubünden.

Die Forschung am SIAF ist darauf ausgerichtet, eine enge Zusammenarbeit mit klinischen Institutionen in Davos, der Universität Zürich sowie weiteren spezialisierten Forschungszentren zu fördern. Das Institut widmet sich einer patientenzentrierten translationalen Forschung mit einem starken Fokus auf die Aufklärung immunologischer Mechanismen allergischer Erkrankungen und von Asthma. Ziel dieser Arbeiten ist die Entwicklung innovativer Präventions- und Therapiestrategien für betroffene Patientinnen und Patienten.
Das SIAF pflegt eine enge Zusammenarbeit mit führenden internationalen Organisationen, darunter die European Academy of Allergy and Clinical Immunology, die American Academy of Allergy, Asthma & Immunology sowie die World Allergy Organization. Darüber hinaus bestehen enge Kooperationen mit renommierten akademischen Institutionen wie dem Sean Parker Center for Allergy and Asthma Research der Stanford University sowie der Harvard T.H. Chan School of Public Health der Harvard University. Das SIAF ist zudem ein Research Center of Excellence der EAACI und der WAO.

Des Weiteren verfügt das Institut über ein wachsendes Team von Bioinformatikerinnen und Bioinformatikern und integriert zunehmend Ansätze der Künstlichen Intelligenz sowie des Machine Learnings in seine Forschung. Mehrere fortgeschrittene Projekte in diesem Bereich befinden sich derzeit in Bearbeitung.

Im Jahr 2025 wurden insgesamt 59 wissenschaftliche Arbeiten in international begutachteten Fachzeitschriften mit Impact Factor publiziert beziehungsweise befinden sich noch im Druck. Das SIAF erreichte dabei einen Gesamt-Impact Factor von 616,6, was einen Durchschnitt von 11,418 Punkten pro Publikation bedeutet. Die neuesten Forschungsergebnisse wurden in 40 Abstracts auf verschiedenen Fachkongressen präsentiert. Mitarbeitende des SIAF erhielten Einladungen zu 98 Seminaren und Vorträgen an nationalen und internationalen Kongressen. Solche Einladungen sind von grosser Bedeutung für die internationale Sichtbarkeit der erzielten Forschungsergebnisse sowie für die Anerkennung der wissenschaftlichen Leistungen des Instituts. SIAF Mitglieder übernahmen 2025 den Vorsitz von 44 wissenschaftlichen Sitzungen und engagieren sich in 67 Funktionen innerhalb internationaler Fachgesellschaften sowie in 34 Positionen bei internationalen Fachzeitschriften. Seit 2018 ist Prof. Dr. Cezmi A. Akdis Chefredaktor der Fachzeitschrift Allergy. Unter seiner Leitung stieg der Impact Factor der Zeitschrift von 6,02 auf 12, womit sie zur weltweit führenden Fachzeitschrift im Bereich Allergologie und Klinische Immunologie wurde. Aufgrund seiner international hoch angesehenen wissenschaftlichen Publikationen wurde Prof. Dr. Cezmi A. Akdis im Jahr 2025 bereits zum zehnten Mal in die Liste der weltweit meistzitierten Forschenden aller wissenschaftlichen Disziplinen von Thomson Reuters Clarivate aufgenommen. Das SIAF hat bislang rund 1’812 wissenschaftliche Beiträge publiziert und zählt weltweit zu den meistzitierten Instituten seines Fachgebiets. Gemäss Web of Science wurden die Publikationen des SIAF insgesamt 45’000-mal zitiert.

Die von Prof. Dr. Cezmi A. Akdis formulierte Epithelbarriere-Theorie erklärt, dass eine Schädigung der epithelialen Barrieren des Körpers, wie der Haut, der Atemwege und der Darmschleimhaut, ein zentraler Faktor bei der Entstehung zahlreicher chronischer Erkrankungen ist. Dazu zählen Allergien, Asthma, Autoimmunerkrankungen, Stoffwechselerkrankungen sowie bestimmte neuropsychiatrische Erkrankungen. Epitheliale Barrieren bilden die erste Verteidigungslinie des Körpers gegenüber Umwelteinflüssen wie Krankheitserregern, Schadstoffen, Allergenen und Toxinen. Sind diese Barrieren intakt, verhindern sie das Eindringen schädlicher Substanzen in tieferliegende Gewebe und somit die Auslösung von Immunreaktionen. Moderne Lebensgewohnheiten, geprägt durch eine erhöhte Exposition gegenüber verarbeiteten Lebensmitteln, Reinigungsmitteln, Mikroplastik, Luftverschmutzung und Antibiotika, können jedoch die Integrität der epithelialen Barrieren beeinträchtigen. Das führt zu einer erhöhten Durchlässigkeit („Leaky Barrier“), wodurch Mikroorganismen und Gifte in tiefere Gewebeschichten gelangen können. In der Folge werden chronische Entzündungen sowie fehlregulierte Immunreaktionen gefördert. Neuere Forschungsergebnisse zeigen, dass gestörte epitheliale Barrieren nicht nur eine Folge, sondern möglicherweise eine primäre Ursache der Krankheitsentstehung sind, insbesondere bei genetisch prädisponierten Personen. Die Theorie betont zudem die entscheidende Rolle des Mikrobioms, das in enger Wechselwirkung mit den epithelialen Oberflächen steht und wesentlich zur Aufrechterhaltung der Immuntoleranz beiträgt. Das Verständnis und der Schutz der epithelialen Barrieren gelten daher als vielversprechender Ansatz für die Prävention von Erkrankungen sowie für die Entwicklung neuer und wirksamerer Therapien in einem breiten Spektrum chronischer Krankheiten.

Gemäss der Epithelbarriere-Theorie können weitverbreitete moderne Umwelteinflüsse wie Reinigungsmittel, Luftverschmutzung und Lebensmittelzusatzstoffe, insbesondere Emulgatoren und Konservierungsstoffe, die Integrität epithelialer Oberflächen beeinträchtigen und zur Entstehung chronisch-entzündlicher sowie immunvermittelter Erkrankungen beitragen. Detergenzien und Reinigungsmittel, die in Haushalten und öffentlichen Räumen häufig verwendet werden, enthalten Tenside und andere chemische Substanzen, welche die epithelialen Schichten der Haut und der Atemwege schädigen können. Wiederholte Exposition schwächt die sogenannten Tight Junctions zwischen den Epithelzellen, wodurch die Durchlässigkeit der Barrieren erhöht wird. Hier wird das Eindringen von Allergenen, Krankheitserregern und Schadstoffen in tieferliegende Gewebe begünstigt. Besonders kritisch zeigt sich die Situation bei Säuglingen sowie bei Sportlerinnen und Sportlern, da diese Gruppen infolge intensiver Hygienemassnahmen beziehungsweise erhöhter Umweltbelastung häufiger mit solchen Subs-

tanzen in Berührung kommen.
Luftverschmutzung, einschliesslich Feinstaubpartikeln (PM2.5 und PM10), Dieselabgasen, Ozon und Stickstoffdioxid, beeinträchtigt nachweislich die epithelialen Barrieren der Atemwege. Diese Schadstoffe verursachen oxidativen Stress und Entzündungen, schädigen die Zellmembranen der Epithelzellen und verändern die Tight-Junction-Proteine. Dadurch steigt nicht nur die Anfälligkeit für Atemwegsinfektionen und Asthma, sondern es können systemische Immunreaktionen ausgelöst werden. Auch Lebensmittelzusatzstoffe wie Emulgatoren, künstliche Süssstoffe und Konservierungsmittel stehen mit einer erhöhten Durchlässigkeit der Darmbarriere in Verbindung. Emulgatoren wie Polysorbat 80 und Carboxymethylcellulose stören die schützende Darmschleimschicht sowie die Zusammensetzung der Darmmikrobiota. Das kann zu chronischen niedriggradigen Entzündungen, einem Ungleichgewicht des Mikrobioms (Dysbiose) und metabolischen Störungen führen. Die geschädigte Darmbarriere erleichtert zudem das Eindringen von Bakterien und Endotoxinen in den Blutkreislauf, was möglicherweise zur Entstehung von Adipositas, entzündlichen Darmerkrankungen und weiteren chronischen Erkrankungen beiträgt. Insgesamt schwächen diese modernen Umwelt- und Lebensstilfaktoren die natürlichen Schutzbarrieren des Körpers, fördern fehlregulierte Immunreaktionen und könnten eine wesentliche Rolle bei der zunehmenden Häufigkeit allergischer, autoimmuner und metabolischer Erkrankungen spielen.

Precision Proteomics Center: Eines der zentralen Projekte des Instituts ist das Zentrum für Präzisionsproteomik, das in Zusammenarbeit mit dem Kanton Graubünden und der Universität Zürich aufgebaut wurde. Seit 2022 wird das Zentrum von Prof. Messner geleitet und hat nationale und internationale Sichtbarkeit erlangt.
Das Zentrum entwickelt und nutzt Proteomik-Technologien, um zentrale Fragen in Immunologie, Allergologie und Präzisionsmedizin zu adressieren. Seine Aktivitäten verbinden Technologieentwicklung mit anwendungsorientierter Forschung. Das übergeordnete Ziel ist es, in enger Zusammenarbeit mit klinischen Partnern und Biobanken verbesserte Diagnostik und Medikamentenentwicklung zu unterstützen.
· Hautbiomarker: Das Zentrum für Präzisionsproteomik verwendet minimalinvasive Tape-Strip-Proben, um Hauterkrankungen direkt auf molekularer Ebene zu untersuchen. Es hat Methoden für die gleichzeitige Messung von Proteinen und Metaboliten entwickelt und einen der bisher größten Proteindatensätze für Haut erstellt.
· Personalisierte Behandlungsstrategien bei Lymphomen: Das Zentrum untersucht Proteinunterschiede zwischen Lymphomtypen und führt ex vivo Medikamentenstudien in Patientenproben durch, um den personalisierten Einsatz bestehender und neuer Therapien zu unterstützen.
· Proteine in seltenen Immunzellen: Die Gruppe von Prof. Messner entwickelt zudem ultrasensitive Methoden zur Analyse des Proteoms einzelner Immunzellen und seltener Immunzellpopulationen, einschließlich allergenspezifischer B-Zellen.
· IgE-Glykosylierung zur Verbesserung der Allergiediagnostik: Weiters werden sensitive Methoden zur Charakterisierung der IgE-Glykosylierung entwickelt. Dadurch entstehen neue Einblicke in Allergiemechanismen sowie potenzielle Marker für Diagnose, Monitoring und therapeutische Intervention.

Molekulare Allergologie: Im Rahmen des DAViS Centers wurde das R-Paket „GeneSelectR“ entwickelt und auf CRAN veröffentlicht. Es ermöglicht die Analyse komplexer RNA-Sequenzierungsdatensätze mithilfe verschiedener Machine-Learning-Methoden sowie die Bewertung der Klassifikationsleistung und biologischen Relevanz der Ergebnisse. Das Paket ist Teil der fortlaufenden Bemühungen, molekulare und klinische Datensätze im Rahmen des ML-SOS-ALL-Projekts zu integrieren. Durch die Bestimmung der relativen Häufigkeit verschiedener SARS-CoV-2-Varianten im Abwasser konnte gezeigt werden, dass sich neue Virusvarianten während internationaler Sportveranstaltungen im Dezember 2021 sowie während des World Economic Forum Annual Meeting im Januar 2023 verbreiteten. Mithilfe gezielter Proteomik in ex vivo differenzierten Th1-Zellen konnten Peptide nachgewiesen werden, für die bereits Hinweise auf eine Translation aus nicht-kodierenden RNAs bestanden. In einem weiteren Projekt wurde mittels gezielter Proteomik nach Allergenproteinen gesucht, die Lebensmittel kontaminieren. Darüber hinaus wurden die Untersuchungen zur Protein-Prenylierung in aktivierten Th1-Zellen fortgesetzt, wobei die bisherigen Vorstellungen zu Zielproteinen und Lokalisationen der Protein-Prenylierung kritisch hinterfragt wurden. Neben dem Ausbau der IT-Infrastruktur wurden weitere Kooperationsprojekte zwischen dem DAViS Center und dem Center for Precision Proteomics durchgeführt. Dazu gehörten ein Projekt zur verbesserten statistischen Analyse massenspektrometrischer Daten angereicherter Proteine sowie ein gemeinsames Projekt mit dem Swiss Institute for Sports Medicine zur statistischen Analyse nicht massenspektrometriebasierter Proteomikdaten.Im Rahmen der gemeinsamen COVID-19-Studie entstand zudem eine Publikation in Zusammenarbeit mit der Fachhochschule Graubünden, dem SIAF sowie der Medizinischen Universität Lodz. Die Studie identifizierte diagnostische Laborparameter und Prädiktoren für schwere COVID-19-Verläufe. Im ML-SOS-ALL-Projekt wurden Daten südafrikanischer Kinder mit und ohne atopische Dermatitis analysiert, um relevante Transkriptlisten zu erstellen. Darüber hinaus wurden die Analysen von Abwasserproben sowie die Sequenzierung von SARS-CoV-2-RNA-Fragmenten fortgeführt und bis zum Abschluss des WEF22 erweitert.

Immunmetabolismus: Das primäre Ziel unserer Forschung ist die Aufklärung der Wechselwirkungen zwischen immunologischen und metabolischen Signalwegen, um neue Biomarker zu identifizieren und therapeutische Zielstrukturen für die Prävention und Behandlung viraler Atemwegsinfektionen, mikrobieller Dysbiosen, allergischer Erkrankungen, Mechanismen der Immuntoleranz sowie heterogener Asthma-Phänotypen zu validieren. Hierfür untersuchen wir patientenbasierte Proben aus Blut, Lunge und Haut unter Einsatz modernster Einzelzell- und Bulk-Sequenzierungstechnologien, räumlicher Transkriptomik, Proteomik und Metabolomik, kombiniert mit fortgeschrittenen bioinformatischen Analysen. Zur weiteren Aufklärung krankheitsrelevanter Mechanismen nutzen wir zudem Geneditierungstechniken, hochdimensionale Durchflusszytometrie, konfokale Bildgebung sowie dynamische metabolische Analysen einschliesslich Einzelzell-Energieprofilierung.
Unsere Forschung konzentriert sich auf folgende Schwerpunkte:
· Immunologie viraler Infektionen und deren Langzeitfolgen: Infektionen mit Rhinoviren, Respiratorischen Synzytialviren (RSV) und

Bericht des Direktors

Prof. Dr. Cezmi A. Akdis

Coronaviren verändern die angeborenen und adaptiven Immunantworten bei Patientinnen und Patienten mit chronischen Atemwegs- und allergischen Erkrankungen. Wir haben abnorme Mechanismen der antiviralen Immunantwort in den Atemwegen sowie kurz- und langfristige immunologische Folgen von Infektionen bei Asthma- und Allergiepatientinnen und -patienten charakterisiert.

· Im Bereich Immunmetabolismus bei Virusinfektionen, Immuntoleranz, Allergien und Asthma arbeiten wir an 1) dem gestörten mitochondrialen Metabolismus des Bronchialepithels, der für beeinträchtigte antivirale Antworten bei Asthma verantwortlich ist, 2) metabolischen Kontrollpunkten bei der Entwicklung humaner pathogener Th2-Zellen und regulatorischer T-Zell-Antworten, 3) der immunmetabolischen Reprogrammierung allergenspezifischer CD4+-T-Zellen während der Allergenimmuntherapie sowie 4) den Auswirkungen von Typ-2-Entzündungen auf den Keratinozyten-Metabolismus bei atopischer Dermatitis.

· Im Forschungsbereich Mikrobiom und Ernährung bei allergischen Erkrankungen untersuchen wir die Auswirkungen mikrobieller Dysbiosen sowie mikrobieller und ernährungsbedingter Metaboliten auf die Immunfunktion von Atemwegsepithelzellen und verschiedenen T-Zell-Subpopulationen, um neue Strategien zur Prävention von Asthma zu entwickeln.

· Trainierte Immunität und Toleranz als neue Ansätze für Biomarker, Prävention und Therapie bei Asthma und Allergien stellen ein neuartiges Konzept einer unspezifischen immunologischen Gedächtnisbildung durch epigenetische und metabolische Veränderungen dar. Wir untersuchen diese Mechanismen sowohl in Atemwegsepithelzellen als auch in T-Zellen und identifizieren gleichzeitig Biomarker dieser neu entdeckten Prozesse im Serum von Allergiepatientinnen und -patienten sowie von Personen, die sich einer Allergenimmuntherapie unterziehen.

B-Zellen-Immunologie: Wie aus den nachstehenden Projekten ersichtlich wird, ist die Forschungsgruppe B-Zell-Immunologie hervorragend mit modernster Technik ausgestattet, um menschliche B-Zellen in Gesundheit und Krankheit zu untersuchen. 25-jähriges In-vivo-Follow-up allergenspezifischer Gedächtnis-B-Zellen beim Menschen: Die Immunantwort auf Bienengiftallergene bei Imkern stellt eines der besten Modelle zur Untersuchung der Toleranz gegenüber hohen Allergendosen dar. Imkerinnen und Imker sowie die Reaktion auf die allergenspezifische Immuntherapie (AIT) bei bienengiftallergischen Personen werden vergleichend untersucht. Charakterisierung von Gedächtnis-B-Zellen, die spezifisch für Milchallergene sind: Unsere vorläufigen Daten zeigen eine unterschiedliche Expression von 30 Genen, die mit der Toleranz gegenüber Nahrungsmittelantigenen zusammenhängen. Diese Gene werden mittels gezielter Proteomik sowie B-Zell-Knock-in- und Knockout-Experimenten eingehend analysiert. Histamin reguliert Immunantworten durch unterschiedliche Expression von H1- und H2-Rezeptoren: Histamin und der Histaminrezeptor 2 (HR2) fördern die Entwicklung peripherer Toleranz während der spezifischen Immuntherapie (SIT) sowie bei hoher Bienengiftexposition bei Imkern. Aufbauend auf diesen umfassenden Vorarbeiten verfolgen wir das Ziel, unsere vorläufigen Daten zur unterschiedlichen Expression von HR1 und HR2 auf verschiedenen B-Zell-Subpopulationen weiter zu untersuchen.
Detaillierte Untersuchung von Immunzellen aus asthma-diskordanten monozygoten Zwillingen: Wir untersuchen zirkulierende T- und B-Zell-Subpopulationen mittels umfassender Einzelzell-Sequenzierung in Kombination mit Einzelzell-Proteomik, um die Mechanismen der Immuntoleranz während der erdnussspezifischen oralen Immuntherapie (OIT) umfassend zu charakterisieren. Die etablierten Methoden zur Untersuchung antigenspezifischer B-Zellen werden dabei auf Erdnussallergene und allergenspezifische B-Zellen angewendet. Zirkulierende nahrungsmittelallergenspezifische Antikörper bei Patientinnen und Patienten mit eosinophiler Ösophagitis: Diese Studie untersucht die zugrunde liegenden Mechanismen der eosinophilen Ösophagitis (EoE) mit Fokus auf antigenspezifische Immunantworten. Unsere Ergebnisse zeigen erhöhte Konzentrationen von IgG, IgG4, IgA1 und IgA2 gegen verschiedene Nahrungsmittelantigene bei EoE-Patientinnen und -Patienten, mit deutlichen Unterschieden zwischen aktiver und inaktiver EoE. Typ-2-polarisierte Gedächtnis-B-Zellen (MBC2s): Diese Studie zeigt, dass die Behandlung pädiatrischer Patientinnen und Patienten mit atopischer Dermatitis (AD) mit Dupilumab die Typ-2-polarisierten Gedächtnis-B-Zellen (MBC2) sowie die Gesamt-IgE-Spiegel signifikant reduziert. Die Ergebnisse deuten darauf hin, dass MBC2 und Gesamt-IgE von der IL-4-Signalübertragung abhängig sind und dass die Hemmung der IL-4- und IL-13-Signalwege durch Dupilumab AD wirksam kontrolliert, indem diese Marker reduziert werden. Durchflusszytometrische Analysen der Isotypen der Immunglobulin-Schwerketten des B-Zell-Rezeptors: Die präzise Detektion der Schwerketten-Isotypen des B-Zell-Rezeptors (BCR) ist entscheidend für die Erforschung der B-Zell-Immunologie. In dieser Studie untersuchten wir den Einfluss verschiedener Fc-Rezeptor(FcR)-Blockierungsreagenzien auf die Genauigkeit der Detektion von BCR-Schwerketten-Isotypen mittels Durchflusszytometrie und entwickelten eine optimierte Färbemethode für BCR-Isotypen. Humane Ascaris-Infektion und IL-10-produzierende B-Zellen: Unsere Studie zeigte dass eine Infektion mit Ascaris lumbricoides mit erhöhten Frequenzen IL-10-produzierender regulatorischer B-Zellen assoziiert ist, was auf eine immunsuppressive Reaktion bei infizierten Personen hindeutet. Darüber hinaus korreliert die Infektion mit reduzierten Konzentrationen zirkulierender ABA-1-spezifischer IgG1- und IgE-Antikörper, was auf eine Modulation der Antikörperantworten hinweist.

Alpine Medical Campus: Das SIAF betreibt in enger Zusammenarbeit mit seinen unabhängigen Partnern CK-CARE, HGK und Cardio-CARE medizinische Spitzenforschung. Die Entwicklung von «Translationale Medizin an Grenzflächen, Graubünden» (TMG) sowie die Lancierung der ersten Förderprogramme stellen wichtige Meilensteine dar. Diagnostik, Forschung und Therapie ergänzen sich auf dem medizinischen Campus in idealer Weise und schaffen wertvolle Synergien, die den Patientinnen und Patienten direkt zugutekommen. Forschungsergebnisse werden rasch in innovative Therapieansätze und Behandlungsmöglichkeiten überführt und ermöglichen so ein umfassendes diagnostisches und therapeutisches Konzept. Darüber hinaus bilden die Ausbildung, Förderung und Weiterbildung von Wissenschaftlerinnen und Wissenschaftlern (MSc, PhD und Postdocs) sowie von medizinischen Fachpersonen zentrale Bestandteile des Leistungsportfolios. Strategisches Ziel des medizinischen Campus ist der Aufbau eines international anerkannten Kompetenzzentrums für die personalisierte Prävention und Behandlung allergischer und kardiovaskulärer Erkrankungen.

Prof. Dr. Cezmi A. Akdis

Wir freuen uns sehr auf eine intensive Zusammenarbeit im Rahmen des neu entstehenden Alpine Innovation Center.

Schweizerisches Forschungsinstitut für Sportmedizin: Seit der Gründung des Schweizerischen Forschungsinstituts für Sportmedizin (SRISM) hat sich unsere Forschungszusammenarbeit kontinuierlich und erfolgreich weiterentwickelt. Im Fokus stehen Untersuchungen an Eishockeyspielerinnen und -spielern, Langläuferinnen und Langläufern, nichtprofessionellen Athletinnen und Athleten sowie sportlich inaktiven Kontrollgruppen. Dabei erforschen wir die Mechanismen häufiger Atemwegsinfektionen und anderer Infektionskrankheiten bei Sportlerinnen und Sportlern sowie die immunologischen Mechanismen des Trainings. Gemeinsam konnten wir bereits die fünfte wissenschaftliche Publikation veröffentlichen. Darüber hinaus laufen mehrere bedeutende Studien zu den Auswirkungen von Training, Immunmetabolismus, Geschlecht, Umweltfaktoren sowie körperlicher Leistungsfähigkeit und Performance.

Ausbildung, Lehre und Kongresse: Das SIAF spielt eine wichtige Rolle in der Ausbildung von Studierenden sowie in der postgradualen Weiterbildung. Gleichzeitig erfüllt das SIAF Lehrverpflichtungen an der Universität Zürich. Diese umfassen verschiedene Vorlesungsstunden im Bereich Biochemie am Biochemischen Institut. Prof. C. A. Akdis ist Mitglied der Medizinischen Fakultät der Universität Zürich mit Promotionsrecht an der Mathematisch-Naturwissenschaftlichen Fakultät sowie Honorarprofessor an der Bezmialem University Istanbul. Prof. C. A. Akdis und Prof. M. Akdis sind zudem Honorarprofessoren am Tungren Hospital der Peking University, an der Bursa-Uludağ University, an der Wuhan University, am Harvard University T.H. Chan Institute of Epidemiology sowie an der Zhejiang University in China. Prof. Christoph Messner, PD Dr. K. Bärenfaller und PD Dr. M. Sokolowska sind Mitglieder des Lehrkörpers der Universität Zürich. PD Dr. K. Bärenfaller verfügt zudem über das Promotionsrecht an der Mathematisch-Naturwissenschaftlichen Fakultät der Universität Zürich.

Bedeutende aktuelle Auszeichnungen: Im Mai 2025 wurden Prof. Dr. C.A. Akdis und Prof. M. Akdis von der renommierten Zhejiang University in China zu Ehrenprofessoren ernannt. Im Oktober 2025 erhielt Prof. C. Akdis eine weitere Ehrenprofessur von einer der beiden führenden Universitäten Chinas, der Peking University. In Anerkennung der aussergewöhnlichen und langjährigen Verdienste von Prof. C.A. Akdis für die EAACI, insbesondere durch seine visionäre Leitung der Fachzeitschrift Allergy, wurde der EAACI Allergy Award in Cezmi Akdis Prize umbenannt.

Klinische Dienstleistungen: Seit 2019 ist das SIAF Anbieter der OLINK-Technologie und bietet akademischen sowie industriellen Kundinnen und Kunden spezialisierte Messungen zur Bestimmung von Biomarkern an, die als Parameter zur Beurteilung von Krankheitsentwicklung und Krankheitsverlauf dienen. Diese Analysen tragen zu einem besseren Verständnis verschiedener Krankheitsmanifestationen und -verläufe bei. Derzeit ist das SIAF das einzige Labor in der Schweiz, das diese spezialisierten Messungen sowohl national als auch international zur Verfügung stellt. Darüber hinaus bietet das SIAF spezielle zellulär-immunologische Untersuchungen für die Hochgebirgsklinik Davos sowie für weitere interessierte Kliniken und niedergelassene Ärztinnen und Ärzte an. Mittels durchflusszytometrischer Analysen (FACS-Analysen) von Blut, bronchoalveolärer Lavage (BAL) sowie weiteren Gewebeflüssigkeiten werden verschiedene Immunzelltypen und deren Subpopulationen hinsichtlich Entwicklung, Verteilung und Aktivierungsstatus untersucht.

World Immune Regulation Meeting: Das 19te World Immune Regulation Meeting (WIRM-XIX) fand vom 11. bis 14. März 2025 als Präsenzkongress im Kongresszentrum Davos statt. Rund 370 Wissenschaftlerinnen und Wissenschaftler aus mehr als 35 Ländern nahmen an der Tagung teil, um aktuelle Fortschritte in der Immunologie zu diskutieren. Das wissenschaftliche Programm umfasste 120 Fachvorträge sowie 185 Abstract-Beiträge und förderte den Austausch zwischen Nachwuchsforschenden und erfahrenen Wissenschaftlerinnen und Wissenschaftlern. Der jährliche Kongress stärkt die internationale wissenschaftliche Zusammenarbeit und den Wissensaustausch im Bereich der Immunologie und unterstützt die Entwicklung neuer therapeutischer Strategien für immunvermittelte Erkrankungen.

Finanzielle Grundlage: Die Ausgaben und Finanzerträge des SIAF haben sich im Vergleich zu den Vorjahren nur geringfügig verändert. Die Grundfinanzierung des Instituts ist derzeit durch die Hauptförderer sichergestellt. Sie besteht hauptsächlich aus Beiträgen des Bundes (Forschungsförderungsgesetz Art. 15), Beiträgen des Kantons Graubünden und der Gemeinde Davos, Beiträgen der Universität Zürich, Beiträgen des Schweizerischen Nationalfonds, den EU-Konsortialprojekten CURE und Syn-Air-G sowie Beiträgen von Stiftungen wie der PROMEDICA-Stiftung und der Stiftung ehemalige Bündner Heilstätte Arosa, welche Doktoratsprogramme unterstützen. Zusätzliche Aufwendungen konnten durch Einnahmen aus kompetitiv eingeworbenen Drittmitteln sowie durch den WIRM-Kongress gedeckt werden.

Danksagung: Ich möchte allen Mitarbeitenden meinen herzlichen Dank für ihre hervorragende Arbeit sowie für die Schaffung eines so positiven und kollegialen Arbeitsumfelds am SIAF aussprechen. Ebenso gilt mein aufrichtiger Dank den Kliniken in Davos, ihren Chefärztinnen und Chefärzten sowie deren Teams und der Universität Zürich für die kontinuierliche und wirkungsvolle Unterstützung unseres Instituts.

Besonders hervorheben möchte ich die fruchtbare Zusammenarbeit mit CK-CARE, die es uns ermöglicht, patientenorientierte Forschung im Bereich der atopischen Dermatitis durchzuführen. Mein besonderer Dank gilt hierbei Frau und Herrn Kühne für ihren grosszügigen Support, der unsere Forschung dabei unterstützt, nachhaltige Lösungen zur verbesserten Diagnose und Behandlung von Patientinnen und Patienten mit atopischer Dermatitis zu entwickeln. Dank dieser Förderung konnten zahlreiche Master- und Doktorabschlüsse am Institut realisiert sowie eine grosse Zahl internationaler Fellowships vergeben werden. Seit der Gründung von CK-CARE wurden im Rahmen dieser äusserst erfolgreichen Initiative mehr als 60 internationale Kurzzeit-Fellowships unterstützt.

Bericht des Direktors

Prof. Dr. Cezmi A. Akdis

Mein besonderer Dank gilt der Stiftung Schweizerisches Forschungsinstitut für Hochgebirgsklima und Medizin (SFI), ihrem Stiftungsrat sowie ihrem Vorstand für die langjährige und kontinuierliche Unterstützung. Brigitta Gadient, der Präsidentin unserer Stiftung, möchte ich meine herzliche Wertschätzung aussprechen und ihr für die Zukunft weiterhin viel Erfolg wünschen.

Nicht zuletzt danke ich dem Kanton Graubünden, dem Staatssekretariat für Bildung, Forschung und Innovation sowie allen öffentlichen Stellen, die der Forschung des SIAF grosses Interesse entgegenbringen und das Institut auf vielfältige Weise fördern.

Davos, Mai 2026

Prof. Dr. Cezmi A. Akdis

The Swiss Institute of Allergy and Asthma Research (SIAF), founded in 1988 by the Medical Department of the Swiss Research Institute for High Altitude Climate and Medicine Davos (SFI), aims to improve understanding of allergies and asthma through its research. Since 1996, SIAF has been affiliated with the University of Zurich and has been a member of the Life Science Zurich Graduate School since 2008, which is a joint education project of the University of Zurich and ETH Zurich. This affiliation allows SIAF to offer comprehensive PhD training. Additionally, SIAF is an active member of Academia Raetica and the Graduate School of the Canton of Graubünden.

Research at SIAF is structured to promote close collaboration with clinical institutions in Davos, the University of Zurich, and other specialized research centers. The institute is dedicated to patient-centered translational research, with a strong focus on uncovering the immunological mechanisms underlying allergic diseases and asthma. These efforts aim to drive the development of innovative preventive and therapeutic strategies for affected individuals.

SIAF actively engages with leading international organizations, including the European Academy of Allergy and Clinical Immunology (EAACI), the American Academy of Allergy, Asthma & Immunology (AAAAI), and the World Allergy Organization (WAO). It also maintains strong collaborative ties with prominent academic institutions, such as Stanford University's Sean Parker Center for Asthma and Allergy Research and Harvard University's T.H. Chan School of Public Health. SIAF is a Research Center of Excellence of the EAACI and World Allergy Organization.
In addition, the institute hosts a growing team of bioinformaticians and is increasingly integrating Artificial Intelligence and Machine Learning approaches into its research, with several advanced projects currently underway.

In 2025, 59 scientific papers were published or are still in press in peer-reviewed international journals with an "Impact Factor." The SIAF achieved a total "Impact Factor" value of 616.6 and an average of 11.418 points per publication in 2025. The latest findings were also presented in 40 abstracts at various professional conferences. Our employees were invited to participate in 98 different seminars and presentations at national and international congresses. Such invitations are important for disseminating the achieved results and for the international acceptance of the institute's research. SIAF employees chaired 44 different sessions. Additionally, SIAF employees hold 67 scientific positions in international societies and 34 positions in international journals. Furthermore, since 2018, Prof. CA. Akdis has held the position of Editor-in-Chief of the journal Allergy. With his leadership the impact factor of Allergy increased from 6.02 to 12, which became the number one journal of the specialty of Allergy and Clinical Immunology, As a result of the internationally highly esteemed scientific publications, Prof. Dr. CA. Akdis was included in Thomson Reuters Clarivate's list of most cited researchers from all scientific disciplines worldwide for the tenth time in 2025. The SIAF has published around 1,812 scientific contributions and is among the most cited institutes worldwide. The articles published by the SIAF have been cited 45'000 times according to Web of Science.

The epithelial barrier theory postulated by Cezmi Akdis proposes that damage to the body's epithelial surfaces—such as the skin, respiratory tract, and gut lining—is a key factor in the development of many chronic diseases, including allergies, asthma, autoimmune disorders, metabolic conditions, and even some neuropsychiatric illnesses. Epithelial barriers serve as the body's frontline defense against environmental insults such as pathogens, pollutants, allergens, and toxins. When intact, these barriers prevent harmful substances from entering underlying tissues and triggering immune responses. However, modern lifestyles—characterized by increased exposure to processed foods, detergents, microplastics, air pollution, and antibiotics—can compromise epithelial integrity. This leads to increased permeability or "leaky" barriers, allowing the translocation of microbes and toxins into deeper tissues, which in turn promotes chronic inflammation and dysregulated immune activation. Emerging research highlights that disrupted epithelial barriers are not merely consequences but may be primary drivers of disease onset, especially in genetically susceptible individuals. The theory also emphasizes the critical role of the microbiome, which interacts closely with epithelial surfaces and contributes to immune tolerance. Understanding and preserving epithelial barrier health is thus seen as a promising strategy for disease prevention and the development of new, more effective therapies in a wide range of conditions.

According to the epithelial barrier theory, common modern exposures such as detergents, air pollution, and food additives—particularly emulsifiers and preservatives—can disrupt the integrity of epithelial surfaces and contribute to the development of chronic inflammatory and immune-mediated diseases. Detergents and cleaning agents, widely used in homes and public spaces, contain surfactants and other chemicals that can damage the skin and respiratory epithelial layers. Frequent exposure weakens tight junctions between epithelial cells, increasing permeability and allowing allergens, pathogens, and toxins to penetrate into deeper tissues. This is especially concerning in infants and athletes, who are more frequently exposed to such substances due to hygiene practices and environmental contact.

Air pollution, including particulate matter (PM2.5 and PM10), diesel exhaust, ozone, and nitrogen dioxide, has been shown to impair respiratory epithelial barriers. These pollutants induce oxidative stress and inflammation, damage epithelial cell membranes, and alter tight junction proteins. This not only increases susceptibility to respiratory infections and asthma but can also trigger systemic immune responses. Food additives, such as emulsifiers, artificial sweeteners, and preservatives, are linked to increased gut permeability. Emulsifiers like polysorbate 80 and carboxymethylcellulose disturb the intestinal mucus layer and microbiota, leading to low-grade inflammation, microbiome imbalance (dysbiosis), and metabolic disturbances. This damage facilitates the translocation of bacteria and endotoxins into circulation, potentially contributing to obesity, inflammatory bowel disease, and other chronic conditions. Together, these modern exposures erode the body's natural protective barriers, promote dysregulated immune responses, and may play a key role in the increasing prevalence of allergic, autoimmune, and metabolic diseases.

Report of the director

Prof. Dr. Cezmi A. Akdis

Precision Proteomics Center: One of the Institute's key projects is the Precision Proteomics Center at SIAF, established in collaboration with the Canton of Grisons and the University of Zurich. Since 2022, the Center has been led by Prof. Messner and has gained national and international visibility.
The group develops and applies proteomics technologies to address key questions in immunology, allergy, and precision medicine. Its activities combine technology development with application-driven research, with the overall aim of supporting improved diagnostics and drug development in close collaboration with clinical partners and biobanks.
· Skin biomarkers: The Center uses minimally invasive tape-strip samples to study skin diseases directly at the molecular level. It has developed workflows for simultaneous protein and metabolite measurements and generated one of the largest molecular skin maps to date.
· Personalized treatment strategies in lymphoma: The Center generates proteomic maps across lymphoma types and performs ex vivo drug perturbation studies in patient samples to support the more personalized use of existing and emerging therapies.
· Proteins in rare immune cells as disease targets: The group of Prof. Messner further develops ultra-sensitive methods to analyze the proteome of single immune cells and rare immune-cell populations, including allergen-specific B Cells.
· IgE glycosylation to improve allergy diagnostics: The Center develops novel sensitive workflows to characterize IgE glycosylation, providing new insights into allergy mechanisms and potential markers for diagnosis, monitoring, and therapeutic intervention.

Molecular Allergology: In the context of the DAViS Center, the GeneSelectR R package was created and made available on CRAN. It allows the analysis of complex RNA sequencing datasets using different machine learning methods and assessing the classification performance and biological relevance of the results. It is part of our continuing efforts to integrate molecular and clinical datasets in the ML-SOS-ALL project. By determining the relative prevalence of different SARS-CoV-2 variants in wastewater, we showed that a new variant spread at international sports events in December 2021 and at the WEF in January 2023. We used targeted proteomics in ex vivo differentiated Th1 cells to detect peptides with previous evidence of translation from non-coding RNAs. In another project, we searched for allergen proteins contaminating food using targeted proteomics.
We continued investigating protein prenylation in activated Th1 cells, challenging the current view on the targets and sites of protein prenylation.In addition to the IT infrastructure installation, there were further projects in collaboration between the DAViS Center and the Center for Precision Proteomics. These included a project for improved statistical analysis of mass spectrometry data of enriched proteins, as well as a project with the Swiss Institute for Sports Medicine (SRISM) for the statistical analysis of non-mass spectrometry-based proteomics data. A publication was made in collaboration with the University of Applied Sciences Graubünden (FHGR), SIAF, and the Medical University of Lodz, Poland, as part of the collaborative COVID-19 study. The study identified diagnostic laboratory parameters and predictors for a severe course of COVID-19. In the ML-SOS-ALL project, data on South African children with and without atopic dermatitis were analyzed to create relevant transcript lists. The analysis of wastewater samples and sequencing of SARS-CoV-2 RNA fragments were continued and were completed by WEF22.

Immunometabolism: The primary objective of our research is to elucidate the interactions between immune and metabolic pathways, with the aim of identifying novel biomarkers and validating therapeutic targets relevant to the prevention and treatment of respiratory viral infections, microbial dysbiosis, allergic diseases, immune tolerance mechanisms, and heterogeneous asthma phenotypes. We investigate patient-derived samples from blood, lungs, and skin using a combination of single-cell and bulk sequencing technologies, spatial transcriptomics, proteomics, and metabolomics, integrated with advanced bioinformatic analyses. To uncover the underlying mechanisms of disease, we further employ gene editing techniques, high-dimensional flow cytometry, confocal imaging, and dynamic metabolic assays, including single-cell energy profiling. Our research focuses on the following topics:
· Immunology of viral infections and their long-term consequences: Infections with rhinoviruses, respiratory syncytial viruses and coronaviruses alter innate and adaptive immune responses in patients with chronic respiratory and allergic diseases. We have characterized abnormal mechanisms to viruses in the airways and short- and long immune consequences of infections in patients with asthma and allergies.
· Immunometabolism in viral infections, immune tolerance, allergy and asthma: We are working on 1) abnormal mitochondrial metabolism of bronchial epithelium responsible for impaired antiviral responses in asthma, 2) metabolic checkpoints for development of human pathogenic Th2 cells and regulatory T cell responses, 3) immunometabolic reprogramming of allergen-specific CD4+T cells during allergen immunotherapy, and 4) effects of type 2 inflammation on keratinocyte metabolism in atopic dermatitis.
· Microbiome and nutrition in allergic diseases: We are investigating the effect of microbial dysbiosis and microbial and nutritional metabolites on immune function of the airway epithelial cells and various T cells subsets in order to establish novel strategies in asthma prevention.
· Trained immunity and tolerance as novel avenues for biomarkers, prevention and treatment in asthma and allergy: It constitutes a novel concept of unspecific immunological memory via epigenetic and metabolic modifications. We have been investigating these mechanisms in airway epithelial cells and in T cells, as well as we identify biomarkers of these newly discovered processes in serum of patients with allergies and undergoing allergen immunotherapy.

B Cell Immunology: As can be seen from the below projects, the B Cell Immunology group is equipped with state-of-the-art technologies to investigate human B Cells in health and disease. 25-year in vivo follow-up of allergen-specific memory B Cells in humans. Immune response to venom allergen in beekeepers represents one of the best models to investigate high dose allergen tolerance. Responses in beekeepers and bee venom–allergic individuals undergoing allergen immunotherapy (AIT) will be investigated comparatively. Characterization of memory B Cells specific for milk allergens: Our preliminary data demonstrated differential expressi-

on of 30 food antigen tolerance-related genes. We will perform in-depth analysis of these genes with targeted proteomics and B Cell knock-in and knockout experiments. Histamine regulates immune responses by differential expression of H1 and H2 receptors: Histamine and Histamine receptor (HR) 2 promote the development of peripheral tolerance during SIT and high dose venom exposure in beekeepers. With this extensive background, we propose to pursue our preliminary data on the differential expression of HR1 and HR2 on B Cell subsets.
In depth investigation of immune cells from asthma-discordant monozygotic twins: We are investigating circulating T and B Cell subsets by extensive single cell sequencing combined with single cell proteomics. We aim to comprehensively characterize the mechanisms of immune tolerance during peanut-specific oral immunotherapy (OIT). Advanced methodologies for investigating antigen-specific B Cells will be applied to peanut allergens and peanut allergen-specific B Cell responses. Circulating food allergen-specific antibodies in eosinophilic esophagitis patients: This study investigates the underlying mechanisms of eosinophilic esophagitis (EoE), focusing on antigen-specific responses. Our findings indicate elevated levels of IgG, IgG4, IgA1, and IgA2 against various food antigens in EoE patients, with notable differences between active and inactive EoE. Type 2 polarized memory B Cells (MBC2s): This study demonstrates that dupilumab treatment in pediatric atopic dermatitis (AD) patients significantly reduces Type 2–polarized memory B Cells (MBC2) and total IgE levels. The findings suggest that MBC2 cells and total IgE levels depend on IL-4 signaling. Dupilumab-mediated inhibition of IL-4 and IL-13 signaling effectively controls AD by reducing these markers. Flow Cytometric Analyses of B Cell Receptor Immunoglobulin Heavy Chain Isotypes: Accurate detection of B Cell receptor (BCR) heavy chain isotypes is critical for studying B Cell immunology. We evaluated the effects of various Fc receptor (FcR) blocking reagents on the accurate detection of BCR heavy-chain isotypes by flow cytometry and developed an optimized staining protocol. Human Ascaris infection and IL-10 producing B Cells: Our study found that Ascaris lumbricoides infection is associated with higher frequencies of IL-10–producing regulatory B cells. Additionally, the infection correlates with reduced levels of circulating ABA-1-specific IgG1 and IgE, indicating a modulation of antibody responses.

Alpine Medical Campus: The SIAF, in close collaboration with its independent partners CK-CARE, HGK and Cardio-CARE, conducts cutting-edge medical research. The establishment of “Translationale Medizin an Grenzflächen, Graubünden” (TMG) and the launch of its first funding initiatives represent important milestones. Diagnostics, research and therapy complement each other ideally on the medical campus, creating valuable synergies that directly benefit patients. Research findings are rapidly translated into innovative therapeutic approaches and treatment options, enabling a comprehensive diagnostic and therapeutic concept. In addition, the education, training and continuing professional development of academics (MSc students, PhD candidates and postdoctoral researchers) as well as medical professionals are key components of the institution’s activities. The strategic goal of the medical campus is to establish an internationally recognized center of excellence for personalized prevention and treatment of allergic and cardiovascular diseases. We are very much looking forward to extensive collaboration within the framework of the newly developing Alpine Innovation Center.

Swiss Research Institute for Sports Medicine: After the establishment of Swiss Research Institute for Sports Medicine (SRISM) our research collaboration has been efficiently growing focusing on ice hockey players, cross-country players, non professional athletes and nonsportive controls investigated the mechanisms of common respiratory infections infectious diseases in athletes and immune mechanisms of training. We achieved the publication of the 5th paper together and several key studies are going on focused on effects of training, immune metabolism, gender, effects of environmental exposure, physical capacity and performance.

Education, teaching, congresses: SIAF plays an important role in the education of students and postgraduate studies. At the same time, SIAF fulfills teaching obligations at the University of Zurich. These obligations include various lecture hours within the framework of biochemistry at the Biochemical Institute. Prof. C. A. Akdis is a faculty member of the Medical Faculty of the University of Zurich with the right to confer doctorates in the Faculty of Mathematics and Natural Sciences and an honorary professor at Bezmialem University Istanbul. Prof. C. A. Akdis and Prof. M. Akdis also hold honorary professorships at Tungren Hospital of Peking University, University of Bursa-Uludag, and Wuhan University, Harvard University T.H. Chan Institute of Epidemiology, Zhejiang University, China. Prof. Christoph Messner, PD Dr. K. Bärenfaller and PD Dr. M. Sokolowska are members of the teaching staff at UZH and PD Dr. K. Bärenfaller holds right to confer doctorates in the Faculty of Mathematics and Natural Sciences of the UZH.

Recent important awards: In May 2025, Prof. Dr. C.A. Akdis and Prof. M. Akdis were awarded honorary professorships by the renowned Zhejiang University in China. In October 2025, Prof. C. Akdis received an additional honorary professorship from one of China’s two leading universities, Peking University. In recognition of Prof. C.A. Akdis’ outstanding and longstanding contributions to EAACI, particularly through his visionary leadership of the journal Allergy, the EAACI Allergy Award was renamed the Cezmi Akdis Prize.

Clinical service: Since 2019, SIAF has been a service provider of OLINK technology, offering its academic and industrial customers special measurements to determine biomarkers that serve as parameters for assessing disease development and monitoring. These measurements also help in better understanding various manifestations and progressions of diseases. Currently, SIAF is the only laboratory in Switzerland offering these specialized measurements for both domestic and international purposes. The SIAF offers special cellular immunological investigations to the Davos clinic and all other interested clinics and practicing physicians. Using flow cytometric analysis (FACS analysis) of blood, bronchoalveolar lavage (BAL), as well as other tissue fluids, the different immune cells and subpopulations are measured in terms of their development, proportions, and activation state.

Report of the director

Prof. Dr. Cezmi A. Akdis

World Immune Regulation Meeting: The 19th World Immune Regulation Meeting took place as an in-person congress at the Davos Congress Center from 11 to 14 March 2025. The meeting gathered approximately 370 scientists from more than 35 countries to discuss recent advances in immunology. The program included 120 scientific presentations and 185 abstract contributions and facilitated exchange between early-career researchers and senior scientists. The congress promoted international scientific collaboration and knowledge exchange in the field of immunology and supported the development of novel therapeutic strategies for immune-mediated diseases.

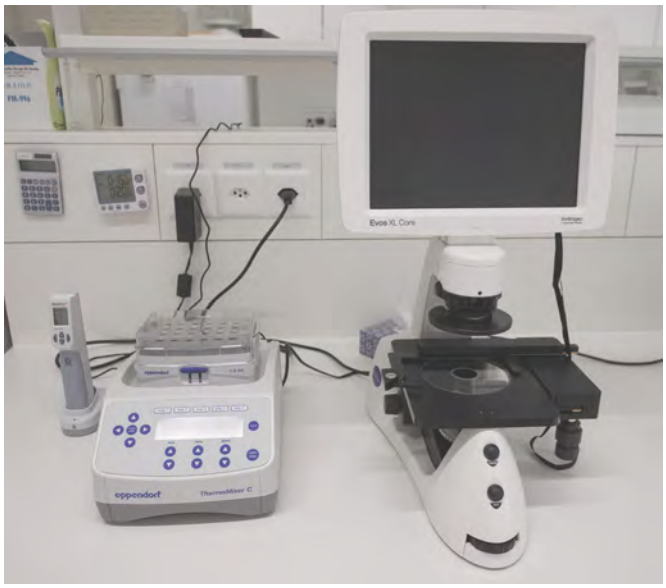
Financial basis: SIAF's expenses and financial income have changed only insignificantly compared to previous years. Basic funding of the Institute is currently ensured by the main sponsors. It consists mainly of a contribution from the federal government (Research Promotion Act Art. 15), contributions from the Canton of Graubünden and the municipality of Davos, contributions from the University of Zurich, contributions from the Swiss National Science Foundation, EU Consortium Grants CURE and Syn-Air-G and contributions from foundations, such as the PROMEDICA Foundation and the Foundation formerly Bündner Heilstätte Arosa, which support doctoral programs. The additional expenses were covered by income from additional competitively acquired third-party funds and the WIRM congress.

Acknowledgements: I would like to express my heartfelt thanks to all staff members for their outstanding work and for fostering such a positive and collaborative working environment at SIAF. My sincere appreciation also goes to the clinics in Davos, their chief physicians, and their teams, as well as to the University of Zurich for their consistent and effective support of our institute.

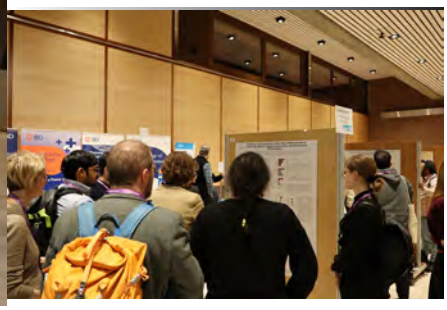
I would particularly like to highlight our fruitful collaboration with CK-CARE, which enables us to conduct patient-oriented research in atopic dermatitis. I am especially grateful to Mrs. and Mr. Kühne for their generous support, which empowers our research to develop sustainable solutions for improved diagnosis and treatment of patients with atopic dermatitis. Thanks to this support, numerous Master's and PhD degrees have been earned at the institute, and a large number of international fellowships have been awarded. This highly successful initiative has supported more than 60 short-term international fellowships since the founding of CK-CARE.

My deep appreciation also goes to the Swiss Research Institute for High Altitude Climate and Medicine Foundation (SFI), its Board of Trustees, and its Executive Committee for their unwavering support. I would like to extend my sincere thanks and best wishes for continued success to Brigitta Gadient, the President of our Foundation.

Last but not least, my special thanks go to the Canton of Graubünden, the State Secretariat for Education, Research and Innovation, and all public authorities who have demonstrated a strong and committed interest in SIAF's research and continue to support the institute in every possible way.
Davos, May 2026



Prof. Dr. Cezmi A. Akdis



World Immune Regulation Meeting

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Prof. Dr. Cezmi A. Akdis

Director

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Ögülür Ismail	PhD, Junior Group Leader **/***,

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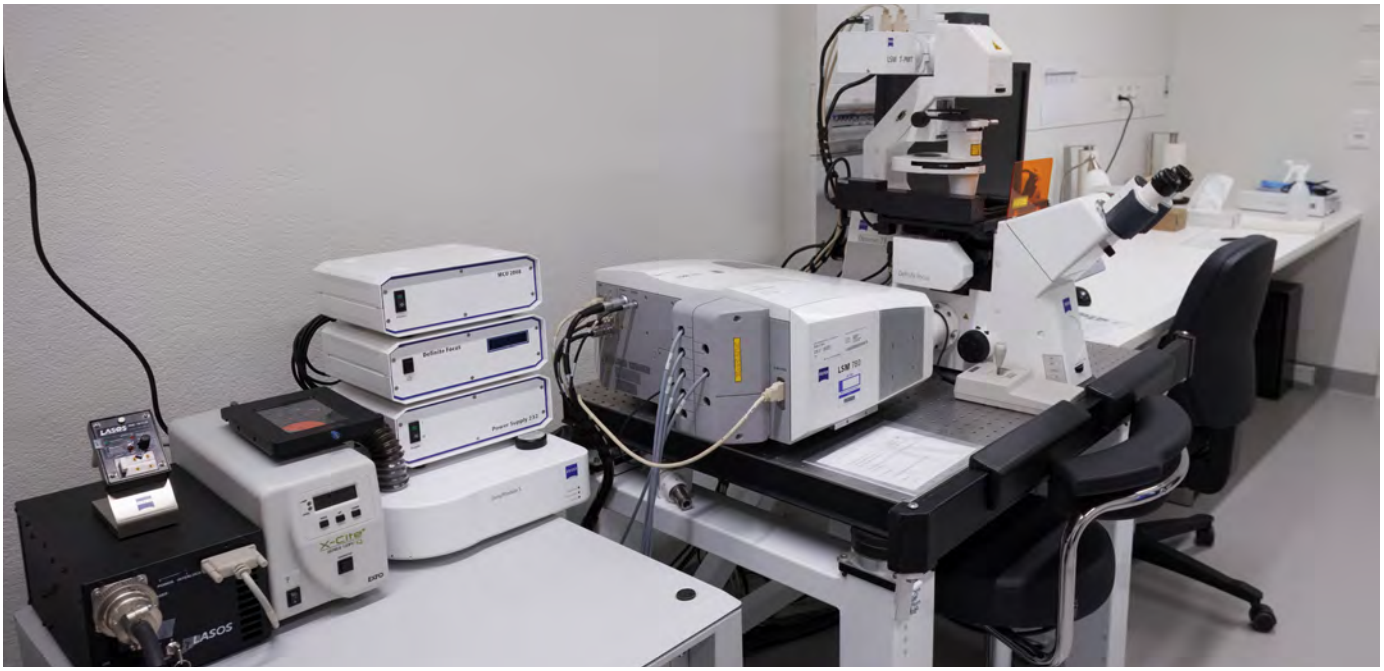
L. Harra, Prof. Dr. C. phys., Direktorin PMOD/WRC, Davos (mit beratender Stimme)
H. Wälti, lic. oec. HSG, Quästor, Bad Ragaz (mit beratender Stimme)



SIAF Members 2025

Prof. Dr. Cezmi A. Akdis

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Ardicli Sena	Acad. guest *** (until 03/25)
Babayev Huseyn	Acad. guest MSc ***
Barletta Elena	PhD student *** (until 12/25)
Bel imam Manal	PhD student *** (until 02/25)
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Bürgi Laura	Technician *
Cachej Carl-Philipp	Acad.guest, *** (07-08/25)
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Kim Juno	Acad. guest, MSc ***
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Stocker Nino	Technician *
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Yang Minling	PhD student ***
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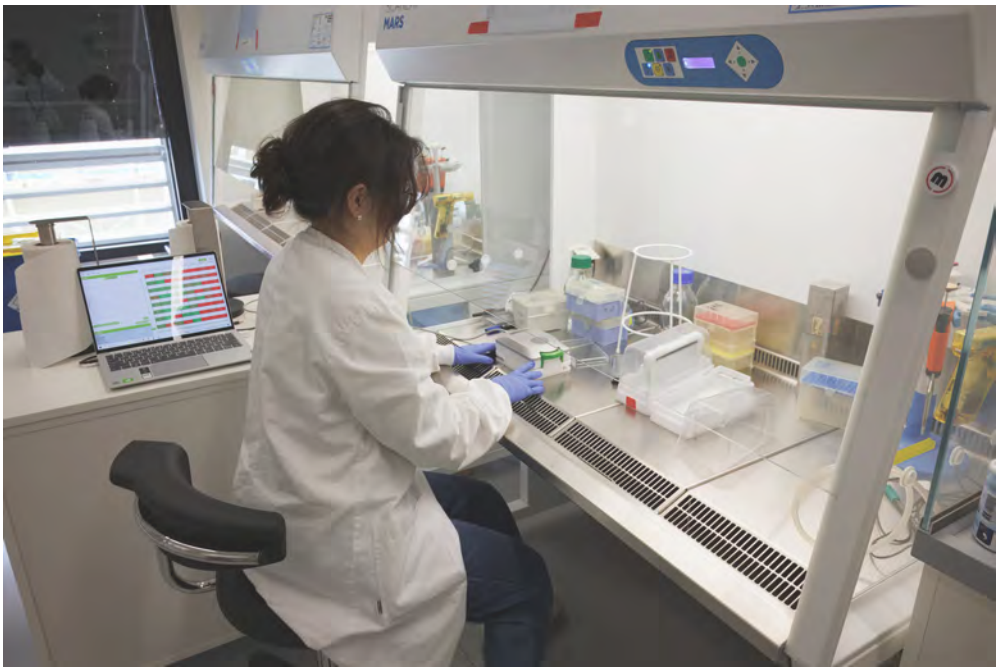


Cellular Allergy / Immunology

Prof. Dr. Cezmi A. Akdis

The epithelial barrier theory: Links to health, longevity and chronic noncommunicable diseases.

Humans are exposed to a variety of toxins and chemicals every day. It is estimated that more than 350'000 new chemicals have been introduced to our lives after the chemistry revolution in 1950s, mostly without any reasonable control of their health effects. Following this a substantial increase in the frequency of allergic, auto immune and neuropsychiatric diseases started in industrialized countries. Many studies are now pointing to the rapid rise of exposure to many of these harmful substances during the last six decades with their implications on skin and mucosal epithelial barrier functions.

Our initial research into the epithelial barrier theory began in 1998, when we delved into intensively studying skin disorders and demonstrated that eczema in atopic and contact dermatitis manifests when T cells in the skin are triggered by allergens, foreign substances, and microbial products (Trautmann et al J Clin Invest 2000, Journal Allergy Clin Immunol 2002. The activated T cells migrate to the epidermis, where they engage with and eliminate keratinocytes. This discovery led us to formulate the epithelial barrier theory in skin health, which we later expanded to encompass the respiratory system, sinuses, and gut. In 2006, an editorial was published proposing a dual role of the epithelial barrier function, as both a first line of physical defense, creating a protective layer, and an immune regulatory role with an expulsion response (Akdis, Current Opinion in Immunology 2006). When internal inflammation reaches a certain threshold, the barrier intercellular junctions open to expel cells and proinflammatory cytokines outward to the lumen away from the affected tissue, mitigating the inflammatory burden. Overall, this process contributes to the chronicity of allergies and other noncommunicable diseases. A few years later, our studies demonstrated a strong immune response around the epithelial barrier against foreign agents, is representing the main molecular mechanism by which tight junctions can open or close in response to certain stimuli. Simultaneously, a pivotal discovery regarding filaggrin mutations in the stratum corneum underscored the clinical significance of restoring the epithelial barrier function. While tight junctions in the skin are located underneath the stratum corneum in the stratum granulosum, they hold an even higher importance in the gut, lungs, and sinuses (Soyka Journal Allergy Clin Immunol 2012), where they serve as the primary line of defense due to the absence of a protective stratum corneum. Around the year 2013, as we understood the role of the immune response and inflammation in regulating epithelial barriers, our focus turned to investigating the impact of various environmental factors on the epithelial barrier function. These included air pollution, particulate matter, microplastics, detergents, household cleaners, and food emulsifiers. What surprised us were the effects we observed on the skin, particularly in their ability to open tight junctions and trigger the release of inflammatory factors. For instance, surfactants such as sodium lauryl sulfate, commonly found in soaps and detergents, were found to alter the surface tension and compromise the integrity of the epithelial barrier (Xian Mu, Journal Allergy Clin Immunol 2015). Our research, which centered on the skin, led us to further validate our findings using human organoids and organs-on-a-chip.

The following substances have been identified to play a role in damaging epithelial barriers, causing microbial dysbiosis and immune system activation. Air pollutants: particulate matter (PM10, PM2.5), ozone, nitrogen oxides, carbon monoxide, sulfur dioxide, diesel exhaust; volatile organic compounds: benzene, toluene, formaldehyde present in cleaning products, paints, and building materials. Detergents and surfactants: chemicals such as sodium lauryl sulfate damage the skin, airway, and gut barriers. Food additives: emulsifiers (e.g. polysorbate 20, polysorbate 80), preservatives, pesticides, enzymes; micro- and nanoplastics; cigarette smoke; chlorine in water and swimming pools. According to the epithelial barrier theory (www.epithelialbarriertheory.com), exposure to many of these above listed substances damage the epithelium on the surface of our skin, lungs and intestine. A defective epithelial barrier has been demonstrated in more than 80 chronic noncommunicable diseases such as asthma, atopic dermatitis, allergic rhinitis, chronic rhinosinusitis, eosinophilic esophagitis, celiac disease, and inflammatory bowel disease. In addition, leakiness of the gut epithelium is also implicated in systemic autoimmune and metabolic conditions such as diabetes, obesity, multiple sclerosis, rheumatoid arthritis, systemic lupus erythematosus, ankylosing spondylitis, and autoimmune hepatitis. Finally, distant inflammatory responses due to a 'leaky gut barrier' and microbiome changes are suspected in Alzheimer's disease, Parkinson's disease, chronic depression and autism spectrum disorders. Our early studies have demonstrated the immune system epithelial cell interactions causing an epithelial barrier defect leading to the development of eczematous diseases, asthma and chronic rhinosinusitis.

The "epithelial barrier theory" proposes that the rise in epithelial barrier damaging agents linked to industrialization, urbanization and modern life underlies the rise in allergic, autoimmune and other chronic conditions. After breaking of the epithelial barriers by various environmental agents, microbiome, which normally floats above the skin and mucosas translocates deeper between and beneath the epithelial barrier. Microbial dysbiosis and decreased biodiversity develop following the colonization of opportunistic pathogens. An "epithelitis" with the activation of inflammasome, release of alarmins and multiple chemokines initiates the immune response and inflammation inside and beneath the epithelium with the aim of an

Prof. Dr. Cezmi A. Akdis

"expulsion response" against the deep translocating opportunistic pathogens, pollutants and toxic substances to clear them in deeper tissues. Accordingly, local inflammation, tissue pathology and remodelling reflecting the disease starts. Cells and cytokines leak to the circulation from the barrier defective tissues, migrate and cause inflammation in target tissues in distant organs. Oxidative stress, endoplasmic reticulum stress, unfolded protein response, various mechanisms causing mitochondrial stress, delay and failures in DNA repair, innate and adaptive immune responses have been identified as key mechanisms of tissue injury and inflammation. The loss of the capacity to heal the disturbed epithelial barrier, because of microbial dysbiosis, chronic tissue inflammation and epigenetic mechanisms lead to the continuum of the epithelial barrier defect, microbial dysbiosis and inflammation as well as the chronicity of the disease. Pets and domestic animals are also substantially affected with the burden of epithelial barrier damaging diseases. Companion animals, especially cats and dogs, share living spaces with humans and are exposed to the same hazardous chemicals present in household cleaners, personal care products, air pollutants, and microplastics. The use of cosmetic products and food additives for pets is increasing and, unfortunately, accompanied by less rigorous safety regulations than those governing human products.

Athletes' high-calorie diet often relies on nutritional supplements that may contain food emulsifiers and other additives that disrupt the epithelial barrier function and cause microbial dysbiosis. The type of the materials used in sports equipment and clothing and their extensive exposure, may increase the inflammatory effects. Elite sport competitions can exert pressure on the immune system and epithelial barriers due to the excessive travel-related stress, sleep disturbances and exposure to bacteria from foreign countries. Here, we review the detrimental impact of toxic agents on epithelial barriers and the microbiome, bringing a new perspective on factors affecting the health and performance of elite athletes and physically active individuals.

In conclusion, the extended epithelial barrier theory proposed by Cezmi Akdis explains the increase in many chronic diseases, including allergic, autoimmune, metabolic, and neuropsychiatric disorders. It proposes that modern environmental factors (pollutants, chemicals, food additives, detergents) damage epithelial barriers in the skin, airways, and gut. Barrier disruption leads to microbial imbalance, immune activation, and chronic inflammation.

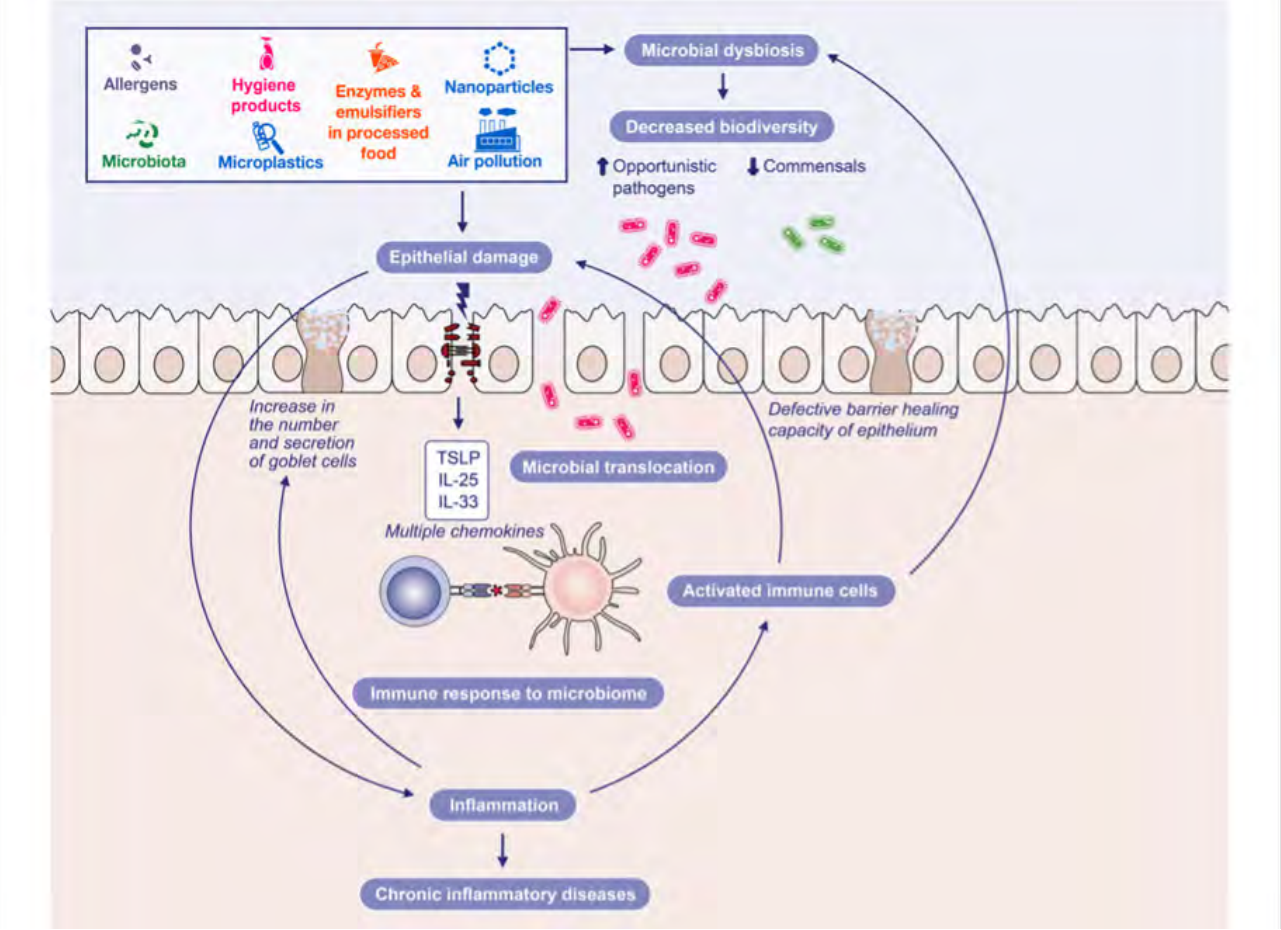


Figure 1. Epithelial barrier damage caused by multiple damaging substances leads to the release of alarmins, microbial dysbiosis and subepithelial infiltration of commensal and dysbiotic microorganisms. The resulting immune response triggers inflammation, which leads to further epithelial barrier damage and the propagation of a vicious cycle. The loss of tolerance to microorganisms, activation of effector immune cells and release of proinflammatory cytokines into the circulation may decrease the threshold/trigger multiple chronic inflammatory diseases at distant sites. FEBS Lett. 2025 Nov;599(22):3208-3243. doi: 10.1002/1873-3468.70113.

Polyethylene Terephthalate Micro and Nanoplastics Induce Oxidative and Endoplasmic Reticulum Stress, Compromising Intestinal Epithelial Integrity and Viability. Sena Ardicli, Huseyn Babayev, Lihong Chang, Ozge Ardicli, Can Zeyneloglu, Yagiz Pat, Duygu Yazici, Xiaoqing Liu, Ezgi Irem Bektas, Carina Beha, Halil I. Akyildiz, Nazrin Babayeva, Muhammed Emin Sari, Beate Rückert, Raja Dhir, Ismail Ogulur, Kari C. Nadeau, Mubeccel Akdis, Cezmi A. Akdis. Allergy doi: 10.1111/all.70291.

Plastic pollution constitutes an escalating global crisis and poses serious threats to ecosystems, human and animal health, food, water and economies. An expanding body of evidence indicates that micro- and nanoplastics cannot be controlled by biological tissue barriers; they can circulate through tissues, reach distant organs, and readily accumulate within various biological systems 1-6. Among the many types of plastics, polyethylene terephthalate (PET) is one of the most common polymers exposed in daily human and animal life. Notably, PET fragments into micro- and nanoparticles that have sparked significant interest regarding potential health implications associated with ingestion.

In the present study, we investigated the effects of PET microplastics on human intestinal cell cultures subjected to a range of concentrations (~200; 1,000; 10,000; 20,000; and 72,000 particles/mL) of PET particles (quantified size range ~6-8 µm, containing a polydisperse mixture of micro- and nanoplastics). Overall, our results delineate the multifaceted nature of microplastic-induced cellular damage, represented by diminished epithelial barrier function, decreased cell viability, and widespread transcriptional shifts converging on oxidative stress, ER stress, and programmed cell death pathways. These in vitro observations contribute to the body of evidence concerning the potential molecular mechanisms of microplastic ingestion to human health. The findings support the continued evaluation and scrutiny of permissible microplastic thresholds in food and water, particularly in the context of increasing global plastic production.

The epithelial barrier theory proposes a comprehensive explanation for the origins of allergic and other chronic noncommunicable diseases. Zeyneloglu C, Babayev H, Ogulur I, Ardicli S, Pat Y, Yazici D, Zhao B, Chang L, Liu X, D'Avino P, Li M, Biçer C, Kurtoğlu Babayev FH, Dhir R, Nadeau KC, Brüggen MC, Akdis M, Akdis CA. FEBS Lett. 2025 Nov;599(22):3208-3243. doi: 10.1002/1873-3468.70113.

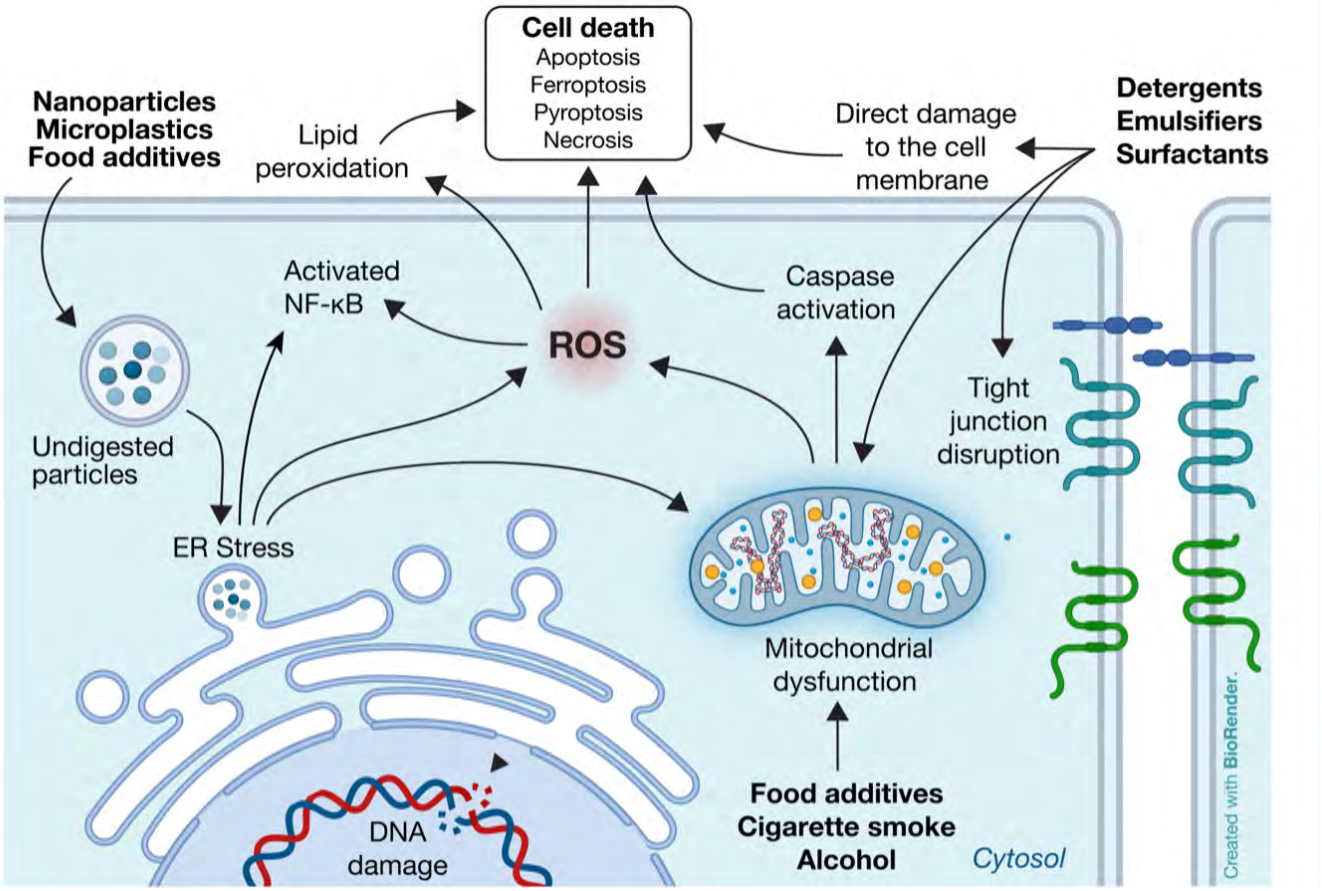


Figure 2. Mechanisms by which environmental toxicants induce cellular damage. Undigested particles (micro- and nanoplastics) trigger endoplasmic reticulum (ER) stress and oxidative stress, leading to NF-κB activation. Alcohol and cigarette smoke cause mitochondrial dysfunction, resulting in reactive oxygen species (ROS) generation and caspase-mediated apoptosis. Surfactants (detergents and emulsifiers) primarily disrupt cellular membranes, causing electrolyte leakage, mitochondrial impairment, tight-junction disruption, and additional ROS production. Excess ROS leads to DNA strand breaks, the release of damage-associated molecular patterns (DAMPs) and NF-κB signaling. If these insults are not resolved, they culminate in multiple forms of cell death—including apoptosis, pyroptosis, ferroptosis, and necrosis. FEBS Lett. 2025 Nov;599(22):3208-3243. doi: 10.1002/1873-3468.70113.

The epithelial barrier theory proposes that modern environmental exposures compromise skin and mucosal surfaces, initiating local inflammation that propagates systemically. The theory integrates epidemiological trends, molecular mechanistic data, and emerging clinical data to show how everyday exposures cause the development and exacerbation of more than 70 chronic noncommunicable diseases. A canonical epithelial cell and barrier injury cascade takes place, generating oxidative stress with increased reactive oxygen species, the release of alarmins, and multiple chemokines and epithelial barrier disruption. The damage to epithelial barriers occurs together with microbial dysbiosis. The alerted immune system fuels various immune activation loops involving multiple cells and chemokines and links barrier leakiness to atopic, autoimmune, metabolic, and neuropsychiatric disease clusters. Supporting the one health concept, abundant exposure of domestic animals and pets to the same groups of toxic substances related to their diseases has recently become more evident. The prevalence of these preventable diseases showed an increase in parallel to industrialization and modernization and epidemiologic exposure to culprit substances throughout the whole world, with a substantial public health burden reaching trillions of dollars each year. Impact statement Modern environmental exposures compromise epithelial barriers, triggering inflammation, microbial dysbiosis, and chronic disease clusters-atopic, autoimmune, metabolic, neuropsychiatric. These preventable conditions now affect millions and impose a public health and economic burden of trillions annually in healthcare costs and lost productivity.

Monosodium Glutamate Induces Cellular Stress, Endoplasmic Reticulum Stress, Mitochondrial Dysfunction, and Cell Death in Intestinal Epithelial Cells. Zhao B, Babayev H, Zeyneloglu C, Pat Y, Yazici D, Ardicli S, García-Sánchez A, Viscardi OG, Akdis M, Nadeau KC, Akdis CA, Ogulur I. Allergy. 2025 Oct;80(10):2916-2920. doi: 10.1111/all.70007.

Monosodium glutamate (MSG), a commonly used food flavor enhancer at recommended levels of 0.1%–1%, has been linked to obesity, metabolic dysregulation, oxidative stress, and potential contributions to irritable bowel symptoms and early-onset rectal cancer. Some research has also shown that glutamate-mediated excitotoxicity is related to autism.5 MSG is sometimes present in foods on its own, while in others it appears in combination with disodium guanylate (DSG) and disodium inosinate (DSI), synergistically enhancing the umami flavor. Although animal studies have demonstrated the effects of these flavor enhancers, data on their impact on human tissues remain limited. Our findings suggest that under conditions of daily exposure, MSG, DSG, DSI, and their combinations can disrupt epithelial barrier integrity, induce different biological processes, including oxidative stress, unfolded protein response and mitochondrial dysfunction, as well as alter key molecular pathways governing autophagy and proteostasis in intestinal epithelial cells. Additionally, our study shows that NAC mitigates the oxidative stress which is induced by 1% MSG in intestinal epithelial cells. Future research will explore antioxidant strategies to reduce MSG-induced cell damage. This work underscores the importance of further research on the molecular mechanisms by which common dietary food additives influence gut barrier function and cellular homeostasis.

The Evolution, Immunopathogenesis and Biomarkers of Type 2 Inflammation in Common Allergic Disorders. Gao YD, Wang ZJ, Ogulur I, Li SJ, Yazici D, Li XH, Pat Y, Zheng Y, Babayev H, Zeyneloglu C, Li YC, Ardicli S, Tian WQ, Ardicli O, Chen SW, Bu XT, Lu G, Chang LH, He Y, Guttman-Yassky E, Cabanillas B, Ozdemir C, Kiykim A, Shamji M, Nadeau K, Torres MJ, Akdis M, Akdis CA.Allergy. 2025 Jul;80(7):1848-1877. doi: 10.1111/all.16620

The prevalence of allergic diseases, including allergic rhinitis, chronic rhinosinusitis, asthma, eosinophilic esophagitis, food and drug allergies, and atopic dermatitis, has been increasing globally over the past few decades. Allergic diseases are closely linked to type 2 immunity, which is characterized by the coordinated interplay between innate and adaptive immune responses. Significant advancements have been achieved in elucidating the cellular and molecular mechanisms that govern type 2 immunity, chiefly mediated by type 2 cytokines, including IL-4, IL-5, IL-9, and IL-13, which are primarily secreted by T helper 2 cells and group 2 innate lymphoid cells. In addition, a diverse array of effector cells, including mast cells, basophils, eosinophils, regulatory T cells, B lymphocytes, dendritic cells, and natural killer cells, are critically involved in orchestrating and modulating type 2 inflammatory responses. The activation of epithelial cells, secretion of alarmins and multiple chemokines, impairment of epithelial barrier integrity, and disruption of microbial dysbiosis serve as crucial mechanisms underlying not only the pathogenesis of allergic disorders but also the development of various systemic conditions. Biologic therapies targeting type 2 pathways-specifically effector functions of IL-4, IL-13, IL-5, thymic stromal lymphopoietin, and immunoglobulin E have-demonstrated promising efficacy. However, a subset of patients with severe allergic diseases remains unresponsive to these treatments, underscoring the need for deeper mechanistic insights and personalized therapeutic approaches. This review addresses the definition, evolution, cellular and molecular basis, and regulation of type 2 immunity. It then examines the common allergic diseases associated with type 2 responses and concludes by exploring the associations between inborn errors of immunity and type 2 responses.

Immune impacts of fire smoke exposure. Johnson MM, Kaushik A, Kline OA, Smith EM, Zhou X, Pat Y, Buergi L, Aguilera J, Alkotob S, Simonin EM, Favaro A, Couto M, Bennett O, Chinthrajah RS, Parsons E, Shamji M, Burke M, Bondy M, Akdis M, Akdis CA, Nadeau KC. Nat Med. 2025 Sep;31(9):3110-3120. doi: 10.1038/s41591-025-03777-6.

Exposure to fire smoke has become a global health concern and is associated with increased morbidity and mortality. There is a lack of understanding of the specific immune mechanisms involved in smoke exposure, with preventive and targeted interventions needed. After exposure to fire smoke, which includes PM2.5, toxic metals and perfluoroalkyl and polyfluoroalkyl substances, epidemiology-based studies have demonstrated increases in respiratory (for example, asthma exacerbation), cardiac (for example, myocar-

dial infarction, arrhythmias), neurological (for example, stroke) and pregnancy-related (for example, low birthweight, premature birth) outcomes. However, mechanistic studies exploring how smoke exposure disrupts cellular homeostasis are lacking. Therefore, we collected blood from smoke-exposed individuals (n = 31) and age-matched and sex-matched non-smoke-exposed controls (n = 29), and investigated these complex interactions using a single-cell exposomic approach based on both methylation and mass cytometry. Overall, our data demonstrated a strong association between smoke exposure and methylation at 133 disease-relevant gene loci, while immunophenotyping showed increased homing and activation biomarkers. We developed an application of mass cytometry to analyze single-cell/metal binding and found, for example, increased levels of mercury in dead cells and cadmium in the live and dead cell populations. Moreover, mercury levels were associated with years of smoke exposure. Several epigenetic sites across multiple chromosomes were associated with individual toxic metal isotopes in single immune cells. Our methods for detecting the effect of smoke exposure at the single-cell level and the study results may help to determine the timing of exposure and identify specific molecular targets that could be modified to prevent and manage exposure to smoke.

The Impact of Rhinovirus, Syncytial Respiratory Virus and Helminth Infection on the Risk of New-Onset Asthma and Other Allergic Conditions-A Systematic Review for the EAACI Guidelines on Environmental Science for Allergic Diseases and Asthma. Agache I, Salazar J, Rodriguez-Tanta Y, Saenz FKF, Haahtela T, Traidl-Hoffmann C, Damialis A, Vecillas L, Giovannini M, Nadeau KC, Pali-Schöll I, Palomares O, Renz H, Schwarze J, Sousa Pinto B, Urrutia-Pereira M, Venter C, Vercelli D, Winders T, Sola-Arnau I, Alonso-Coello P, Canelo-Aybar C, Jutel M, Akdis CA.Allergy. 2025 Jul;80(7):1878-1898. doi: 10.1111/all.16611.

This systematic review evaluated the association between lower respiratory tract infections (LRTI) in infancy with respiratory syncytial virus (RSV), rhinovirus (RV) or infestation with helminths and the risk of developing asthma and allergic diseases. The risk of bias was assessed with ROBINS-E, and the certainty of evidence (CoE) with GRADE. Meta-analysis applied a random-effects model. RSV LRTI is likely associated with an increased risk of developing asthma by age 7 (OR 3.02, 95% CI 2.23-4.09; I2 = 98%; moderate CoE). The impact on wheezing, atopic dermatitis (AD), and allergic rhinitis is uncertain. RV LRTI may be associated with increased risk of developing asthma (OR 8.40, 95% CI 2.56-27.55; I2 = 43%; low CoE). The impact on wheezing and AD is uncertain. Trichuris trichiura infestation might be associated with reduced risk of new-onset wheezing (OR 0.57, 95% CI 0.35-0.94; very low CoE) or AD (HR: 0.35, 95% CI 0.18-0.67; very low CoE). The association between Ascaris lumbricoides and hookworm infestation and the risk of developing asthma or AD is uncertain. Infestation with any helminths might be associated with reduced risk of new-onset asthma by age 5 (OR: 0.60, 95% CI 0.38-0.95; very low CoE) and wheezing (OR 0.70, 95% CI 0.51-0.95; very low CoE). More high-quality studies are needed to confirm these findings.

Aerosol Measurements and Decadal Changes: The Role of Climatic Changes and How It Reflects in Respiratory Allergies and Asthma. Kazadzis S, Fountoulakis I, Damialis A, Masoom A, Papachristopoulou K, Gilles S, Coen MC, Tummon F, Crouzy B, Clot B, Pat Y, Brüggen MC, Nyeki S, Raptis IP, Solomos S, Gkikas A, Moustaka A, Kouremeti N, Akdis CA.Allergy. 2025 Jun;80(6):1613-1628. doi: 10.1111/all.16602.

The causative agents of respiratory allergies are bioaerosols, such as house dust mite feces, pollen grains, and fungal spores. Climate change and urbanization are considered to lead to an increase in the load of allergenic bioaerosols due to impacts on plant phenophases and allergenicity. Continuous and efficient monitoring of the atmospheric composition worldwide is essential, given the major changes involved and their impact on climate change. The complexity of the exposome, evolving from single to multiple complex exposures, is explored in this work. Acquiring information from interdisciplinary scientific disciplines, such as aerobiology (for airborne particles of biological origin), aerosol science (for airborne particles of chemical or inorganic material), and integrating this with the actual reactome of patients with respiratory diseases, we aim to provide evidence of the multifactorial nature of this interaction in real life. The objective of this review is to present how we can monitor aerosols and mostly monitor the exposome, especially the biological one, i.e., pollen and fungal spores, and what their impact is, or could be, on respiratory allergies. A huge technological advancement has been required, as traditional methods of particle collection and identification have been based on tedious laboratory procedures, with delays of more than a week. This has limited their practical use to allergic patients and their treating physicians. Automation, real-time high temporal resolution, and the use of artificial intelligence are being increasingly used in medicine. Likewise, this overview summarizes the current aerosol measurement and modeling capabilities and discusses the classification of various aerosol particles and their impact on respiratory allergies. Satellite remote sensing is highlighted as a solution to the gaps in global aerosol representation by examining aerosol load in the atmospheric column in major cities worldwide. We also discuss potential novel threats, such as pioneer bioaerosols and the respiratory epithelial barrier, as well as future insights into the impact of climate change on allergy and asthma. We conclude with a discussion of emerging co-exposures and co-diseases resulting from the ongoing climate change.

Davos May, 2026

Prof. Dr. Mübeccel Akdis

Immune Regulation

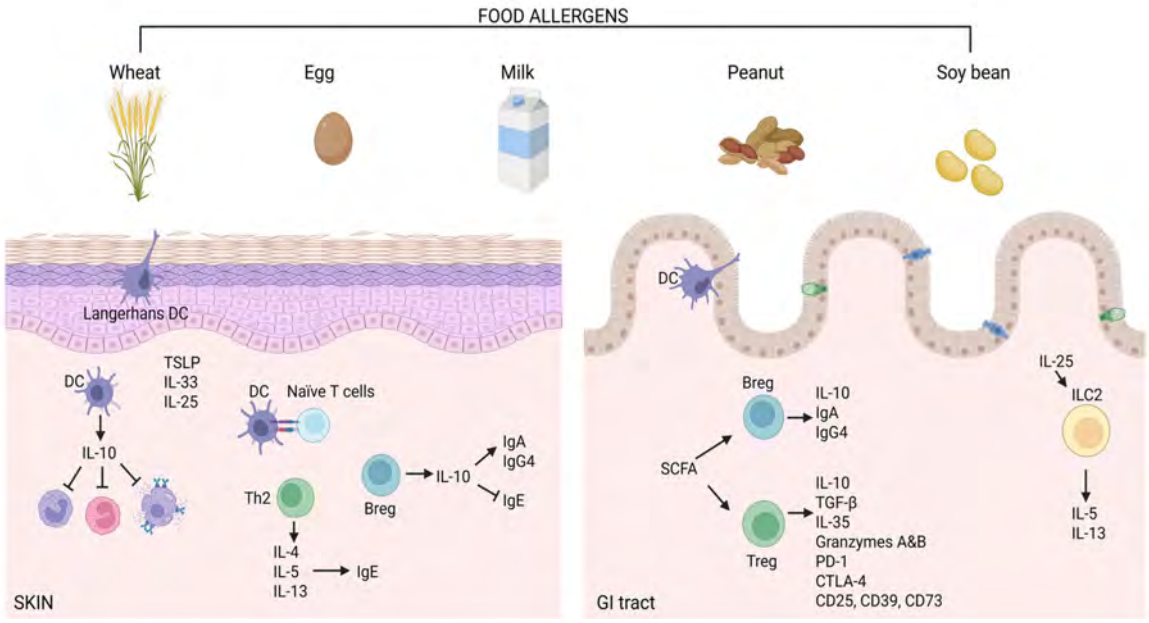
Mechanisms of IgE-mediated food allergy and the role of allergen-specific B Cells

Allergy to food components is a major public health concern and represents a significant challenge in immunology and clinical practice, affecting approximately 7–10% (adults: 10.8% and children ~ 8%) of the population, often leading to severe impacts on the quality of life. It has been defined as an immunological reaction, mediated or not by the immunoglobulin E (IgE) molecule, after exposure to specific food antigens.

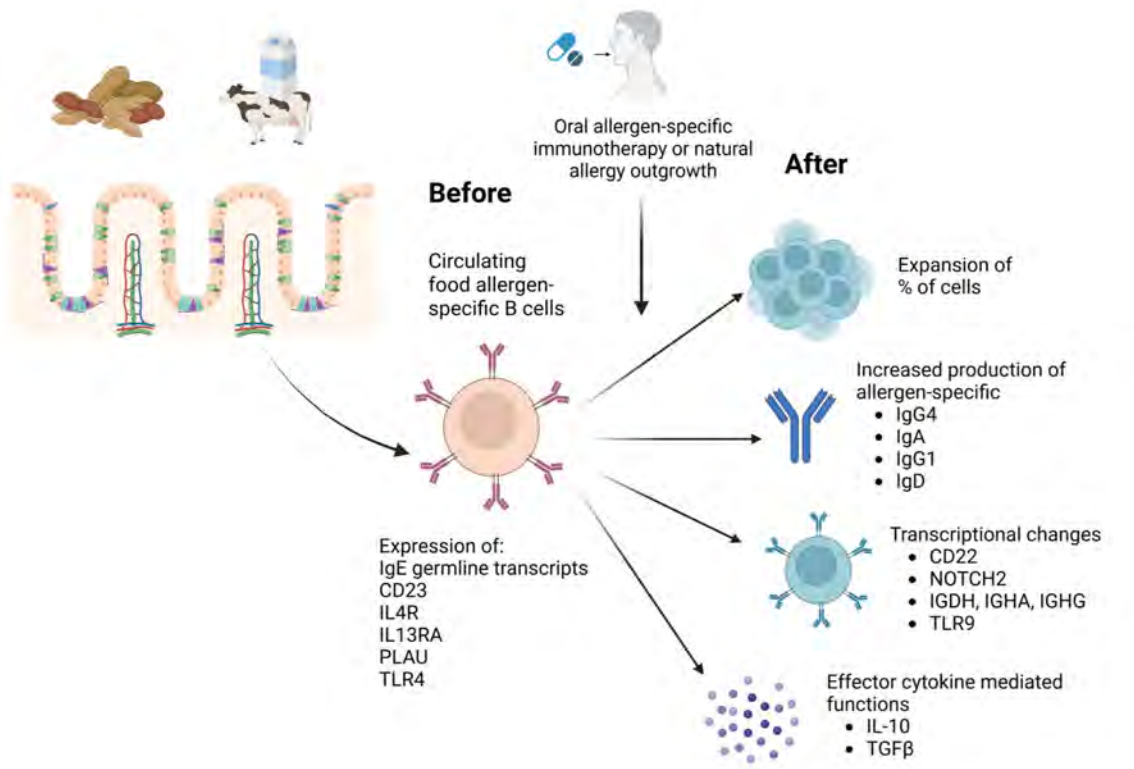
Clinically, these reactions can range from mild symptoms, such as discomfort or itching, to severe and potentially life-threatening responses (anaphylaxis). Additionally, constant dietary exposure requires that the gastrointestinal (GI) tract serve as a tolerogenic organ, but the mechanisms behind this process are not fully understood. Factors such as epithelial barrier integrity, gut flora composition, and immune system players influence whether food antigens trigger an allergic response or are tolerated.

Most common immune-mediated mechanism driving the pathogenesis of food allergies is IgE-mediated hypersensitivity. Upon initial sensitization, allergen-specific IgE antibodies are produced by B Cells, which pass through a process of class switch recombination (CSR), regulated by cytokines such as interleukin (IL)-4 and IL-13. These antibodies bind to high-affinity FcεRI receptors on the surface of mast cells and basophils, and upon cross-linking with a specific allergen, initiate cellular degranulation, resulting in the release of histamine and other proinflammatory mediators. This cascade results in the rapid onset of allergic symptoms (Figure 1).

However, B Cells are not solely involved in promoting allergic reactions through antibody production; they can also play a role in tolerance induction through humoral and cytokine-mediated effector mechanisms. Despite advances in understanding the mechanisms underlying food allergies, many aspects remain poorly understood, especially the role of allergen-specific B Cells in the pathogenesis and development of tolerance (Figure 2).



Different mechanisms of food allergy in skin and gastrointestinal tract. The distinct immunological pathways triggered by food allergens such as wheat, egg, milk, peanut, and soybean in the skin (left) and the gastrointestinal (GI) tract (right). In the skin, allergens activate Langerhans cells and dermal dendritic cells (DCs), promoting the release of epithelial cytokines (TSLP, IL-33, IL-25) that favor Th2 polarization. Th2 cells secrete IL-4, IL-5, and IL-13, driving IgE production and allergic sensitization. Regulatory B Cells (Bregs) can counteract this by releasing IL-10, which supports class switching to IgA and IgG4 while suppressing IgE. In the GI tract, dietary antigens are taken by intestinal DCs, which contribute to the induction of Bregs and regulatory T cells (Tregs), especially in the presence of microbiota-derived short-chain fatty acids (SCFAs). Bregs produce IL-10, IgA, and IgG4, while Tregs mediate tolerance through IL-10, TGF-β, IL-35, granzymes A and B, and inhibitory receptors (PD-1, CTLA-4) and markers (CD25, CD39, CD73). IL-25 can also activate ILC2s, which promote Th2 cytokine production (IL-5, IL-13).



Changes induced in allergen-specific B Cells after allergen-specific immunotherapy and natural allergy outgrowth. Few studies have described features related to allergen-specific B Cell in allergic individuals, mainly the association with the expression of IgE germline transcripts and the expression of CD23 and IL4R. After allergen-specific immunotherapy or in natural allergy outgrowth, cells experience changes in their frequencies, transcriptional profile, and effector functions.

Role of IgG4 Antibodies in Human Health and Disease

IgG4 was at first considered a minor and relatively unimportant subclass of immunoglobulin, as it only constitutes approximately 5% of total serum IgG. However, with advancements in immunological techniques and a growing body of clinical and experimental studies, it has become evident that IgG4 plays a complex and multifaceted role in humans but not in mice, as IgG4 does not exist in mice. Although IgG4 responses play a protective role by mediating immune tolerance to environmental antigens and reducing inflammation during parasitic infections, they can also contribute to pathology in autoimmune diseases and antitumor settings. Evolving as a mechanism to mitigate tissue damage and adapt to chronic exposure to high-dose antigens, IgG4 functions as a paramount component in the immune system's balance between tolerance and overactivation. In health, IgG4 is involved in maintaining immune homeostasis. It is thought to participate in immune tolerance mechanisms, potentially modulating the immune response to self-antigens and harmless environmental antigens. For example, in the context of mucosal immunity, IgG4 may contribute to the regulation of responses against commensal bacteria and dietary antigens, preventing excessive inflammation and tissue damage.

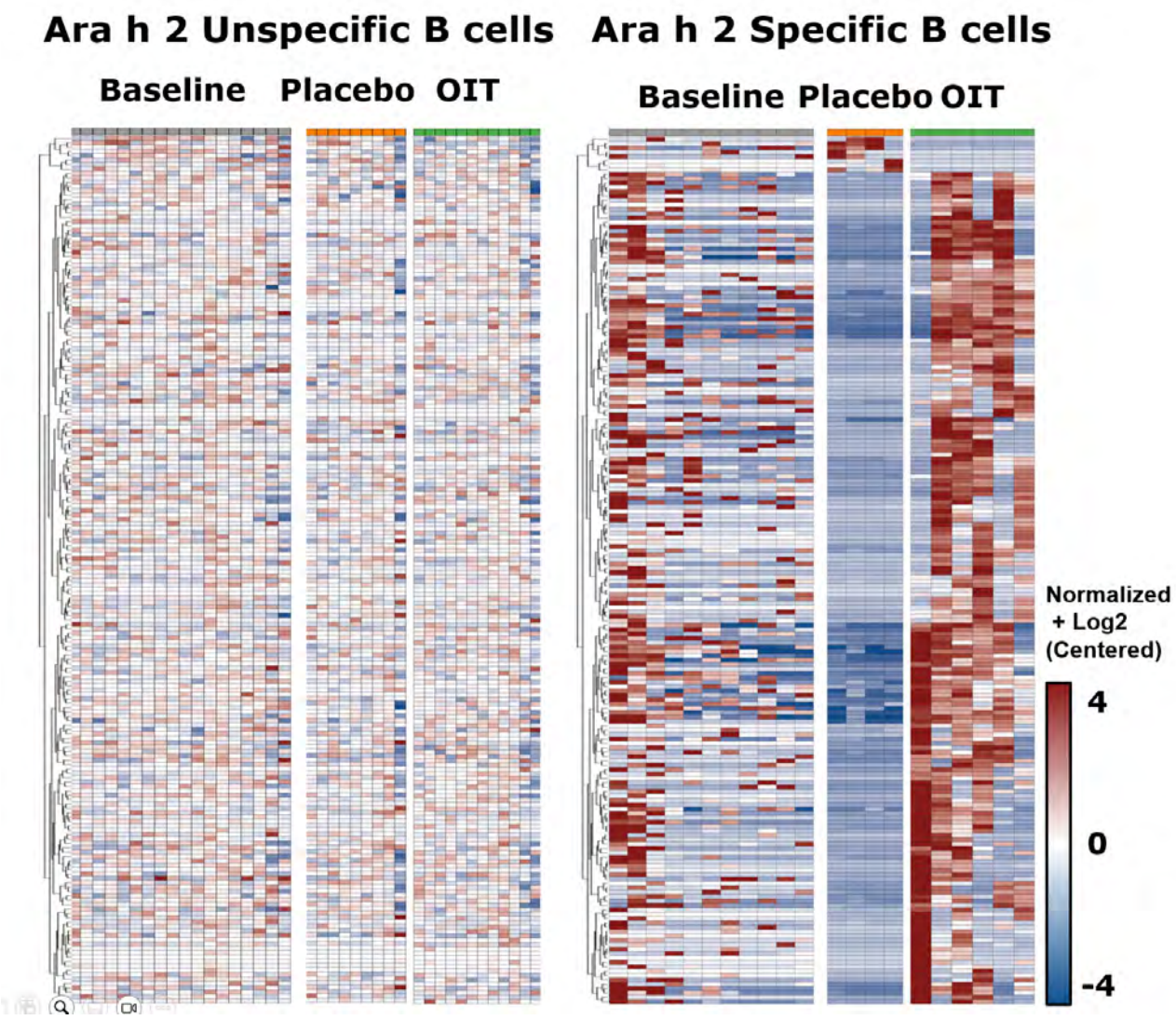
IgG4 plays a critical role in modulating allergic responses and promoting natural tolerance to allergens through unique mechanisms (Figure 2). The induction of IgG4 is heavily reliant on IL-10, as evidenced by its association with high-dose antigen exposure models, acquired natural tolerance, and conditions such as bee venom tolerance and helminth infections. These scenarios highlight the dual role of IgG4, namely, mitigating excessive type 2 responses while contributing to infection persistence or allergen tolerance. Furthermore, elevated IgG4 levels are observed in various contexts, from prolonged exposure to animal allergens to the natural resolution of food allergies, such as cow's milk allergy. In conclusion, IgG4 antibodies are a double-edged sword in human health and disease. In health, they contribute to immune tolerance and homeostasis via Fab-arm exchange, preventing overactive immune responses. However, in diseases, their role is highly complex. In IgG4-related diseases, abnormal IgG4 elevation leads to tissue damage and fibrosis, yet our understanding of the precise mechanisms remains incomplete. In infectious diseases, while they can signify past exposure, they also complicate antibody-based diagnostics. In cancer, IgG4 is potentially involved in tumor immune evasion. Despite these insights, the precise mechanisms driving IgG4-mediated tolerance, particularly in different allergenic contexts, remain an area for further investigation. Understanding these pathways could offer new perspectives on managing allergic diseases and promoting tolerance. For IgG4-related diseases, the focus should be on developing tar-

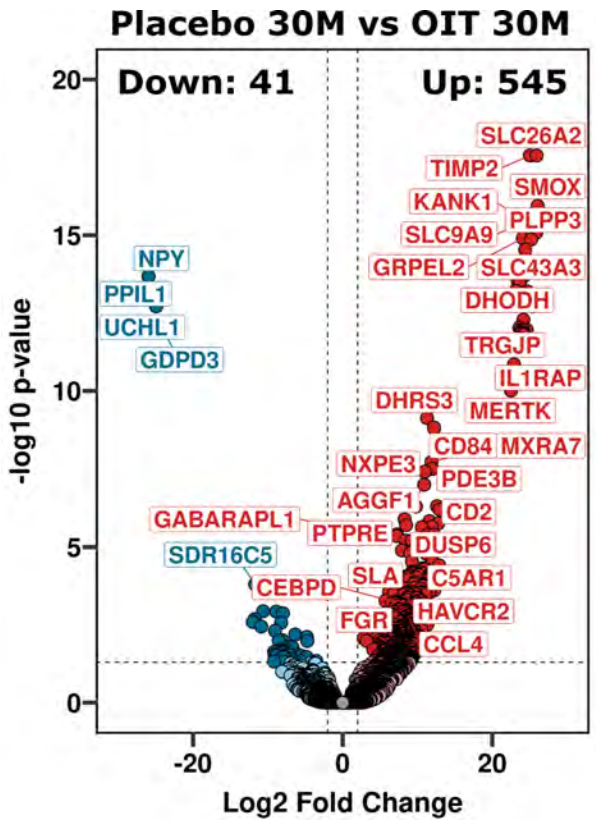
geted therapies to correct the dysregulated IgG4 response. Understanding the regulatory factors of IgG4 in different diseases will uncover novel therapeutic targets.

Allergen-Specific B Cell Tolerance during OIT

Food allergies have become more frequent in the past two decades and are a major public health concern and quality of life burden. The hypersensitivity of food allergies can be remedied by Oral Immunotherapy (OIT), through a continuous low dose exposure to the allergen. While the optimal procedure for OIT is still being discussed and improved, the overall viability of allergen immunotherapy to induce tolerance, has been firmly established in clinical practice. However, the mechanisms behind the changes in allergen reactivity of B Cells, are unclear. Therefore, we aimed to characterize the changed gene expression patterns in allergen-specific B Cells after OIT. We examined peanut major allergen

Ara h 2-specific B Cells and their dynamic changes during 30 months of oral immunotherapy (OIT) followed by 6 months of avoidance. Peanut OIT changes allergen-specific memory B Cells to exhibit genes of regulatory nature and have a greater propensity to communicate with other innate immune cells. Allergen-specific B Cells from allergic participants have upregulated pathways that suggest increased B Cell activation. These B Cells are either already activated or easier to activate by stimulus of allergen exposure. These data further the question how OIT influences B Cells. Instead of changing the B Cell activation threshold, OIT may result in allergen-specific B Cells that behave more like antigen-presenting cells. A shift from an inflammatory B Cell to antigen-presenting B Cell is interesting as it suggests the possibility that the OIT modulates a whole (Figure 3). Findings from studies that analysed changes in peanut-reactive T cells and whole blood transcriptomics support systemic regulatory effects in peanut OIT.





Figuer 3+4: Characterization of Ara h 2-specific B Cells in the OIT and placebo groups after 30 months.

Allergen-specific B Cell responses in oral immunotherapy-induced desensitization, remission, and natural outgrowth in cow's milk allergy.
(Satitsuksanoa P, van de Veen W, Tan G, Lopez JF, Wirz O, Jansen K, Sokolowska M, Mirer D, Globinska A, Boonpiyathad T, Schneider SR, Barletta E, Spits H, Chang I, Babayev H, Tahralı İ, Deniz G, Yücel EÖ, Kıykım A, Boyd SD, Akdis CA, Nadeau K, Akdis M. Allergy. 2025 Jan;80(1):161-180. doi: 10.1111/all.16220. Epub 2024 Jul 11. PMID: 38989779)

Antigen-specific memory B Cells play a key role in the induction of desensitization and remission to food allergens in oral immunotherapy and in the development of natural tolerance (NT). Here, we characterized the role of milk allergen αS1-casein-specific B Cells in desensitization and remission induced by oral allergen-specific immunotherapy (OIT) and in children spontaneously outgrowing cow's milk allergy due to natural tolerance. Increased frequency of circulating milk allergen αS1-casein -specific B Cells was observed after OIT and NT. Milk desensitized subjects showed partial acquisition of phenotypic features of remission, suggesting that desensitization is an earlier stage of remission. Within these most significantly expressed genes, IL10RA and TGFB3 were highly expressed in desensitized OIT patients. In both the remission and desensitized groups, B Cell activation-, Breg cells-, BCR signaling and differentiation-related genes are activated. In NT, pathways associated with innate immunity characteristics, development of marginal zone B Cells and a more established suppressor function of B Cells prevail that may play a role in long-term tolerance. The analyses of immunoglobulin heavy chain genes in specific B Cells demonstrated that IgG2 in desensitization, IgG1, IgA1, IgA2, IgG4 and IgD in remission, and IgD in NT were predominating. Secreted proteins from allergen-specific B Cells revealed higher levels of regulatory cytokines, IL-10 and TGF-β after OIT and NT. Taken together, allergen-specific B Cells are essential elements in regulating food allergy towards remission in OIT-received and naturally resolved individuals.

Allergy discordant twins do not exhibit differences in gene expression in non-switched and switched B Cells.
(Schneider S, Satitsuksanoa P, Babayev H, van de Veen W, Chang I, Yang M, Akdis CA, Nadeau K, Akdis M. Allergy. 2025 Jan;80(1):342-345. doi: 10.1111/all.16376. Epub 2024 Oct 29. PMID: 39470621 No abstract available.)

Davos, May 2026

Prof. Dr. Christoph Messner, PhD

Background

With the decision of the Government of the Canton of Graubünden in 2020, the Swiss Institute of Allergy and Asthma Research (SIAF) was commissioned to establish and operate the Proteomics Center Davos. Since 2022, the Center has been led by Christoph Messner. It has become an integral part of SIAF, with national and international visibility, and acts both as a technology platform and as a research group.

On the one hand, the Center provides state-of-the-art proteomics technologies and services to SIAF, institutions within the Canton of Graubünden, and the University of Zurich (UZH).

On the other hand, it operates as a research group conducting pioneering work in high-throughput and sensitive proteomics to address key mechanistic and translational questions with direct relevance for patients in allergy, immunology, and cancer.

Approximately 50% of its work is dedicated to technology development and the establishment of state-of-the-art workflows, while the remaining 50% focuses on application-driven projects in the field. The application areas align with the Canton's defined goals and SIAF's mission in translational biomedical research and precision medicine, particularly in allergy and inflammatory diseases.

Skin Proteomics for Inflammatory Diseases

Inflammatory skin diseases such as atopic dermatitis are influenced by complex interactions between genetic and environmental factors. To understand these interactions, molecular profiles of the skin need to be studied across large cohorts. However, such studies are limited by the availability of sample material, particularly from skin biopsies.
To overcome this limitation, we established a workflow to profile proteins from skin tape strips, a minimally invasive sampling method. Together with national and international clinical partners and biobanks, we have access to more than 2,000 tape-strip samples, including highly annotated cohorts.
This allows us to associate skin protein profiles with disease activity, barrier function, genotype, and environmental factors, improving our understanding of these diseases and moving us closer to objective, quantifiable tools for diagnosis and therapy monitoring. Our analysis shows how the skin is remodeled in atopic dermatitis by deconvoluting cell-type-associated signatures, connecting cytokines to functional modules, and identifying novel associations with genetic and environmental factors.

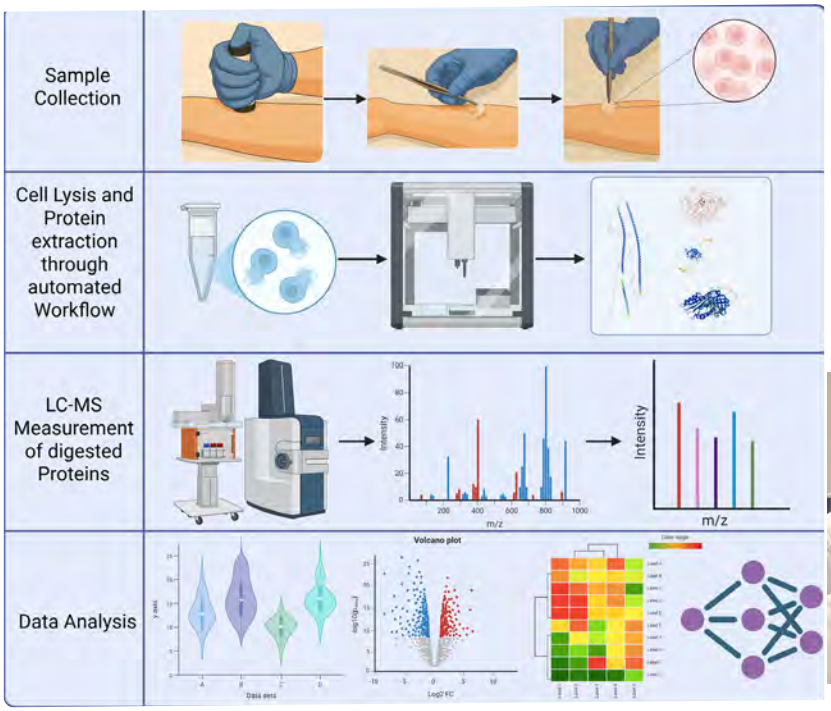
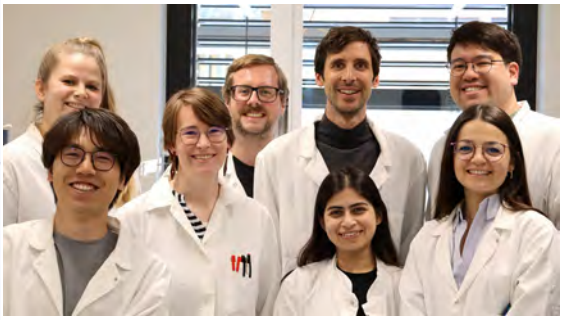


Figure: Overview of the workflow for the analysis of skin tape strips. Created with BioRender.



2025 Group photo Proteomics Center

Precision Proteomics in Lymphoma Research

Lymphomas are a highly heterogeneous group of cancers, with more than 80 distinct subtypes showing different clinical behaviour and treatment responses. This complexity is a major challenge for diagnosis and therapy. Current diagnostic approaches primarily rely on morphology, immunophenotyping, and genetics. However, with more therapeutic options becoming available, a deeper understanding of disease mechanisms and more personalized treatment strategies are urgently needed.
In collaboration with the University Hospital Zurich (Thorsten Zenz and Marco Bühler), we have made significant progress within our LOOP Zurich-funded project to enhance lymphoma diagnostics through proteomics.

Over the past year, we have started processing and analyzing a well-annotated cohort of more than 2,500 tissue and liquid biopsy samples, representing a broad spectrum of lymphoma subtypes and, to our knowledge, the largest proteomics dataset on lymphomas to date.
We have completed a dataset for mantle cell lymphoma, including tissue samples and blood cells. These data show that a single proteomics measurement can capture many routinely used diagnostic markers, as well as features linked to aggressiveness, outcome, and previously defined lymphoma signatures. This highlights the potential of proteomics for diagnostic applications and the rich information content obtained from a single proteomics measurement, which would otherwise require several different technologies.

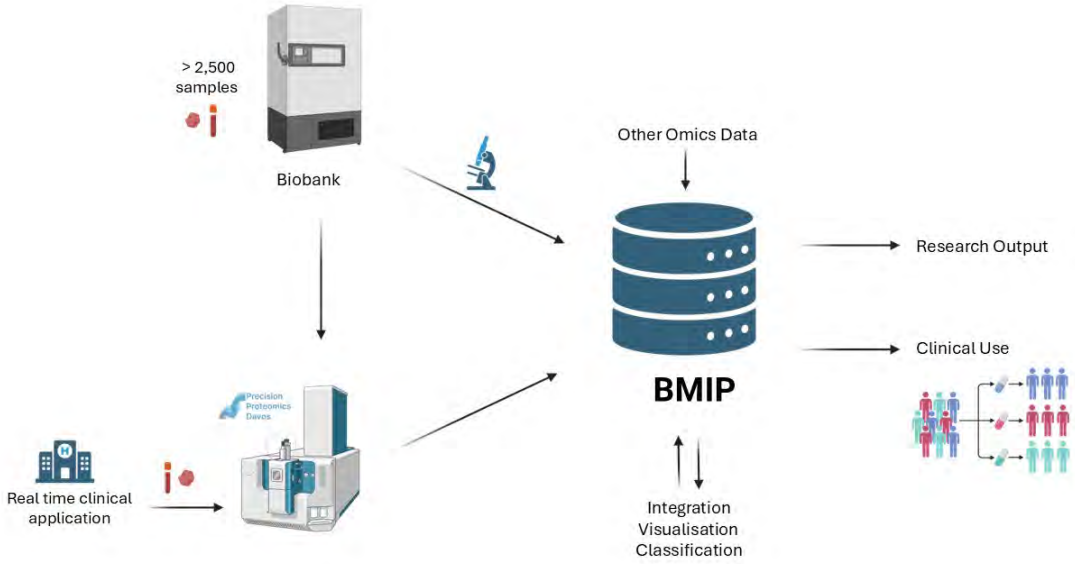


Figure:
Large-scale analysis of lymphoma samples for research and clinical applications.
Created with BioRender.

Characterising the proteome of sorted cells and single cells.

Immune and epithelial cells are highly heterogeneous and therefore require technologies that go beyond bulk measurements for their characterization in disease contexts. While immune cells have been extensively studied using single-cell RNA sequencing, most knowledge remains at the RNA level. In contrast, the functional proteomic landscape is far less explored, and to our knowledge, only a limited number of studies have investigated B Ccell or T-cell proteomes at the single-cell level.
To address this gap, we established workflows for low-input proteomics to analyze rare cell populations and even single cells. Using these workflows, we have already characterized allergen-specific B Cells from as few as 25 cells and identified significant differences linked to a type 2 B Cell phenotype.
We also performed single-cell proteomics on B Cells, showing that known cell types and antibody isotypes can be distinguished at the single-cell level. This provides an important proof of concept for using proteomics to directly characterize rare immune cell populations and single cells. Since proteins are the direct functional readout of cells and the targets of most drug candidates, these workflows provide a basis to identify new targets linked to type 2 B Cell phenotypes.

Glycosylation in Allergies and Immune Regulation

Protein function goes beyond abundance. In particular, post-translational modifications such as glycosylation are key to understanding protein function. This is especially relevant in allergy and immunology, where many key molecules, including cell-surface receptors and antibodies, are highly glycosylated.

One focus of our group has been the characterization of IgE glycosylation, as IgE antibodies are central to allergic responses. However, due to their high degree of glycosylation and low abundance, IgE antibodies are particularly challenging to study.

We established a low-input platform and applied novel fragmentation methods, in particular EAD fragmentation, to identify new forms of glycosylation on IgE that had not previously been detected. The achieved sensitivity now allows us to study IgE glycosylation directly in patient material, which we are currently applying in a large cohort of patient samples.

Mapping the proteomic response to the BTK degrader NX-5948 in primary CLL cells

Chatterjee L, Do THL, Kummer S, Westermann P, Lu J, Zenz T, Messner CB (submitted)

Bruton's tyrosine kinase (BTK) is a central mediator of B Cell receptor (BCR) signaling and a key therapeutic target in chronic lymphocytic leukemia (CLL). BTK inhibitors such as ibrutinib have transformed treatment, but over time, resistance mutations, incomplete target suppression, and off-target toxicities emerge. Targeted protein degraders offer a promising alternative by eliminating BTK protein. Here, we established a high-throughput proteomics workflow in primary patient samples, profiling responses across more than 6,500 proteins and mapping the temporal and dose-dependent effects of BTK degradation using the clinical BTK degrader NX-5948 compared with BTK inhibition by ibrutinib in CLL. NX-5948 induced rapid and consistent BTK degradation, whereas ibrutinib did not significantly alter BTK abundance. Despite this mechanistic difference at the target level, both drugs elicited highly convergent downstream proteomic effects, prominently impacting BCR signaling. In addition, NX-5948 specifically reduced the abundance of five additional kinases — BLK, CSK, GAK, PTK2B, and LCK — in a dose-dependent manner.

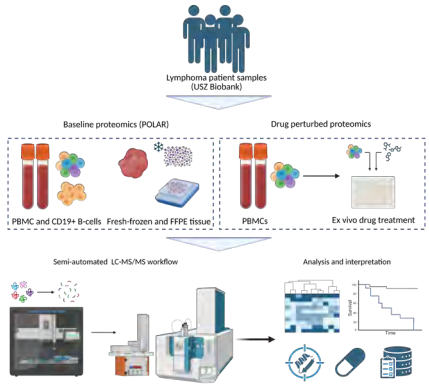


Figure: Overview of the workflow for the analysis of skin tape strips. Created with BioRender.

Serum Proteomics Reveals Survival-Associated Biomarkers in Pancreatic Cancer Patients Treated with Chemoimmunotherapy

Tognetti M, Chatterjee M, Beaton N, Sklodowski K, Bruderer R, Reiter L, Messner CB
iScience. 2025

Immunotherapy has transformed the landscape of cancer treatment but remains largely ineffective for patients with pancreatic ductal adenocarcinoma (PDAC). Some patients, however, show improved outcomes when treated with a combination of immunotherapy and chemotherapy. Here, we conducted deep serum proteome analysis to investigate the protein profiles of PDAC patients and changes during this combinatorial treatment. Utilizing an advanced serum workflow, we quantified 1,011 proteins across 211 samples from 62 patients. Glycolytic enzymes were associated with survival in anti-PD-1 treated patients, with their abundances significantly correlating with expression levels in tumor biopsies. Notably, a set of protein biomarkers was found to be highly predictive of survival in anti-PD-1-treated patients (AUC = 0.91). Overall, our data demonstrate the potential of deep serum proteomics for precision medicine, offering a powerful tool to guide patient selection for treatment through minimally invasive serum protein biomarker measurements.

Preprocessing Large-Scale Proteomics Data

Sun X, Ralser M, Messner CB (accepted, Methods in Molecular Biology)

Recent advances in mass spectrometry instrumentation and computational analysis have established label-free data-independent acquisition (DIA) as a powerful tool in quantitative proteomics, offering depth, throughput, and reproducibility. These developments now enable large-scale projects with hundreds or even thousands of samples, opening new applications in systems biology and biomedicine. However, large-scale datasets require special consideration during data preprocessing. Here, we present a practical guide for preprocessing large-scale DIA datasets, illustrated using a public dataset. We cover key steps, including database searching, normalization, batch correction, missing value imputation, and evaluation of preprocessing strategies, and provide R code together with practical considerations.

Tuft cell IL-17RB restrains IL-25 bioavailability and reveals context-dependent ILC2 hypoproliferation

Feng X, Andersson T, Flüchter P, Gschwend J, Berest I, Muff JL, Lechner A, Gondrand A, Westermann P, Brander N, Carchidi D, De Tenorio KC, Pan T, Boehm U, Klose CSN, Artis D, Messner CB, Leinders-Zufall T, Zufall F, Schneider C

The tuft cell–group 2 innate lymphoid cell (ILC2) circuit orchestrates rapid type 2 responses upon detecting microbially derived succinate and luminal helminths. Our findings delineate key mechanistic steps involving IP3R2 engagement and Ca2+ flux, governing interleukin-25 (IL-25) production by tuft cells triggered by succinate detection. While IL-17RB has a pivotal intrinsic role in ILC2 activation, it exerts a regulatory function in tuft cells. Tuft cells exhibit constitutive Il25 expression, placing them in an anticipatory state that facilitates rapid production of IL-25 protein for ILC2 activation. Tuft cell IL-17RB is crucial for restraining IL-25 bioavailability, preventing excessive tonic ILC2 stimulation due to basal Il25 expression. Supraoptimal ILC2 stimulation by IL-25 resulting from tuft cell Il17rb deficiency or prolonged succinate exposure induces a state of hypoproliferation in ILC2s, also observed in chronic helminth infection. Our study offers critical insights into the regulatory dynamics of IL-25 in this circuit, highlighting the delicate tuning required for responses to diverse luminal states.

Davos, May 2026

Molecular Allergology

PD Dr. Katja Baerenfaller

Molecular Allergology

Exploring T helper 1 cell differentiation and activation

On 3 June 2025, Jana Koch successfully defended her thesis entitled "Differentially Regulated Translation Events in T Helper Cell Differentiation". Her thesis comprised two main aspects important for T helper cell differentiation: 1) investigation of translation and transcription in T helper (Th)1 cells and 2) the prenylation pathway in Th1 cells. The two projects focused on different aspects of Th1 cell differentiation and activation, as our studies on translational regulation showed that transcripts associated with annotated prenylated proteins and proteins in the prenylation pathway were translationally regulated during Th1 cell activation.

The paper summarising the data on the prenylation project, entitled "Uncovering protein prenylation in Th1 cells: novel prenylation sites and insights into statin and farnesyltransferase inhibition", was published in July 2025 in BMC Biology (DOI: doi.org/10.1186/s12915-025-02345-1). The key findings of the study were the identification of novel prenylation sites, both at protein C-termini for geranylgeranylated and particularly farnesylated proteins, as well as at internal sites, and the link between prenylation and the expression of non-canonical protein isoforms. For identified prenylated proteins lacking C-terminal prenylation motifs or cysteines in their canonical isoforms, we searched for the presence of such motifs or cysteines in alternative isoforms and identified isoforms with C-termini containing potential prenylation sites. These isoforms and internal prenylation sites are currently a focus of our ongoing research.

The investigation of protein translation in Th1 cells has meanwhile led to the study of short open reading frame (sORF)-encoded peptides (SEPs). Following up on the translation reads, we built a SEP-specific database containing predicted SEP sequences and used it to search our mass spectrometry data from Th1 cells. As such searches typically suffer from high local false discovery rates, we subsequently followed up the most promising candidates using targeted mass spectrometry with parallel reaction monitoring (PRM). For some SEPs, including SEP_114, full-length peptide sequences were synthesised and used as reference peptides in the PRM measurements to verify the identity of the detected peptides. For SEP_114, the 3D structure predicted by ColabFold furthermore features a high-confidence α-helix, suggesting that it acts as a monomer (Figure 1). These results are currently summarised in a manuscript and form the basis for follow-up projects investigating the potential functions of SEPs with evidence for translation from the translatomics data in Th1 cells (Koch *et al., manuscript in preparation).*

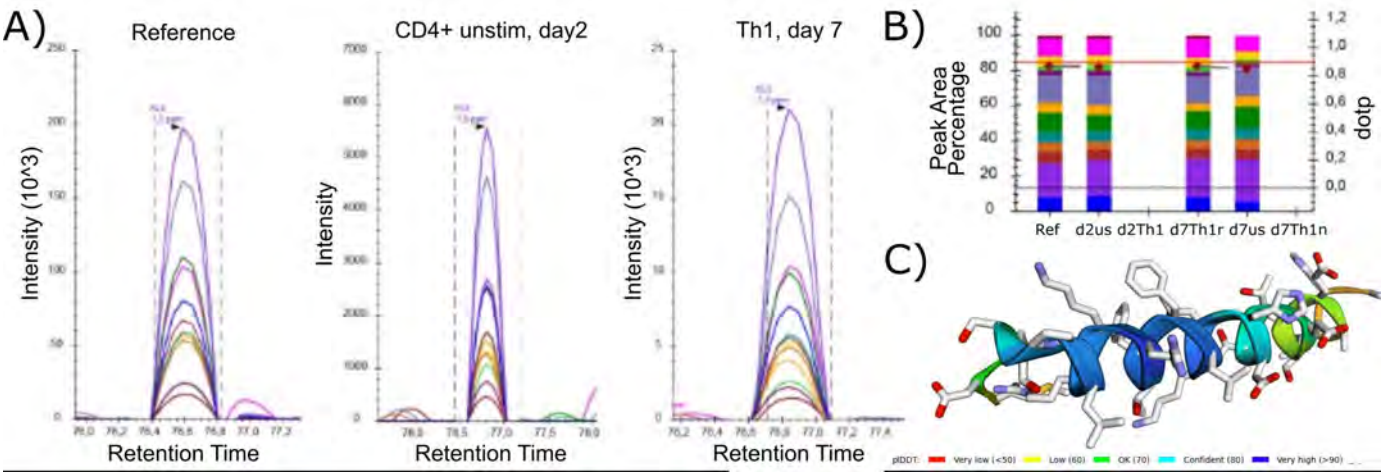


Figure 1: A) Skyline chromatograms showing overlaid fragment ion peaks, and B) peak area graph of the tryptic peptide from the synthetically produced full-length SEP_114 (Reference) and the endogenous peptide in different samples as indicated. C) 3D structure of the SEP_114 as predicted by ColabFold.

Clinical presentation and risk factors for prosthesis-related hypersensitivity in patients with implanted prostheses

In collaboration with Tamara El Saadany and the group of Marie-Charlotte Brüggen at the University Hospital Zurich, we performed a bioinformatic analysis of data from patients with implanted prostheses, with and without prosthesis-related hypersensitivities (PRHs). Based on the results of patch tests, the patients were categorised according to the presence or absence of positive reactions against various metals that are components of prostheses. A second classification was based on the presence or absence of any prosthesis-relevant substance allergy.

Our research questions were whether there were significant associations between the presence of a metal- or prosthesis-relevant allergy and other patient characteristics. These included first clinical features and post-operative symptom dynamics, second allergic co-morbidities, and third the use of medications.

After considerable work on curating and pre-processing the clinical data, we found no significant associations between prosthesis-related hypersensitivity reactions (PRHRs) and various post-operative symptoms, meaning that neither cutaneous nor joint-related symptoms, patterns, or timing of symptom onset were more relevant among PRHR patients. In addition, patients diagnosed with a PRHR to metals or prosthesis-relevant substances did not show any significant associations with pre-existing allergic contact eczema or other allergic co-morbidities, nor with the use of medications including immunosuppressants, corticosteroids, analgesics, antihistamines, or other prescribed drugs (Figure 2).
Although no specific clinical features associated with PRHR could be identified, these findings reinforce the notion that PRH is a relevant issue in prosthesis patients. They support the use of hypoallergenic implants in patients with known contact allergies and highlight the need for future studies aimed at better understanding the underlying pathomechanisms and different types of PRH.

3 June 2025, Defense Jana Koch

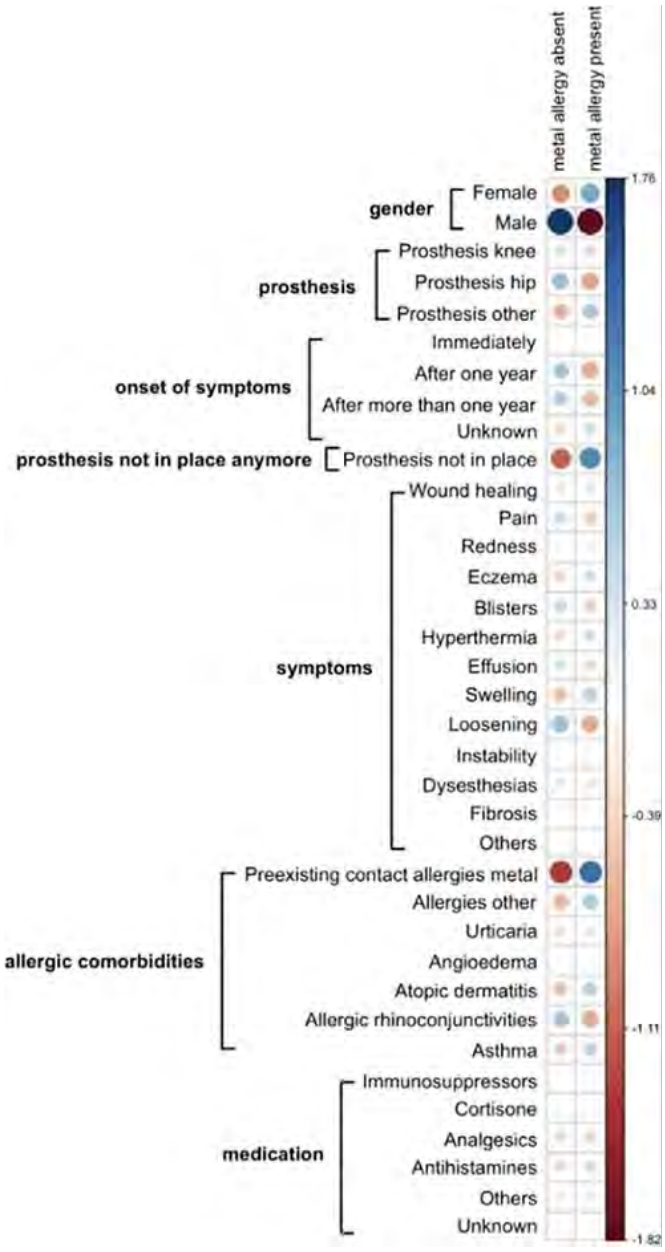


Figure 2: Fig. 4 in El Saadany et al., Clinical Presentation and Causes of Prosthesis-Related Hypersensitivity Reactions: An Analysis of 225 Patients, Int Arch Allergy Immunol 2026; 187:299-307, DOI: DOI: 10.1159/000547103. A chi-square test was used to assess associations between a positive entry in symptoms, symptom onset dynamics, allergic comorbidities, medication use, and the categorization of patients into those with and without a prosthesis-related metal allergy according to the results of the PT. Blue points indicate positive associations, while red points indicate negative associations.

Unlocking Molecular Insights into Sports Medicine using Proteomics

In collaboration with the Swiss Research Institute for Sports Medicine (SRISM), we investigated various aspects of sports medicine in the context of the PhD projects of Elena Barletta using a combination of targeted and non-targeted mass spectrometry-based proteomics across different cohorts.
In the first cohort, differences in the serum proteome of non-athletes were compared with those of amateur athletes and elite athletes, including cross-country skiers and ice hockey players (Figure 3). Analysis and integration of the data showed that cross-country skiers in particular reported higher infection rates.

They also largely shared a distinct immune-related proteomic profile with the non-athletes. Interestingly, and also relevant for the promotion of public health, we found that athletes display protein signatures consistent with beneficial cardiovascular and metabolic adaptations, as well as enrichment of proteins associated with neuronal processes and synaptic organisation. Our findings therefore highlight the complex effects of exercise on systemic physiology and provide novel insights into the molecular basis of exercise-induced adaptations (Barletta et al., submitted).
In the second sports-related project, we analyse total serum and serum extracellular vesicles from female and male elite ice hockey players to gain molecular insights into training and recovery (Barletta, Rüegg *et al., manuscript in preparation).*

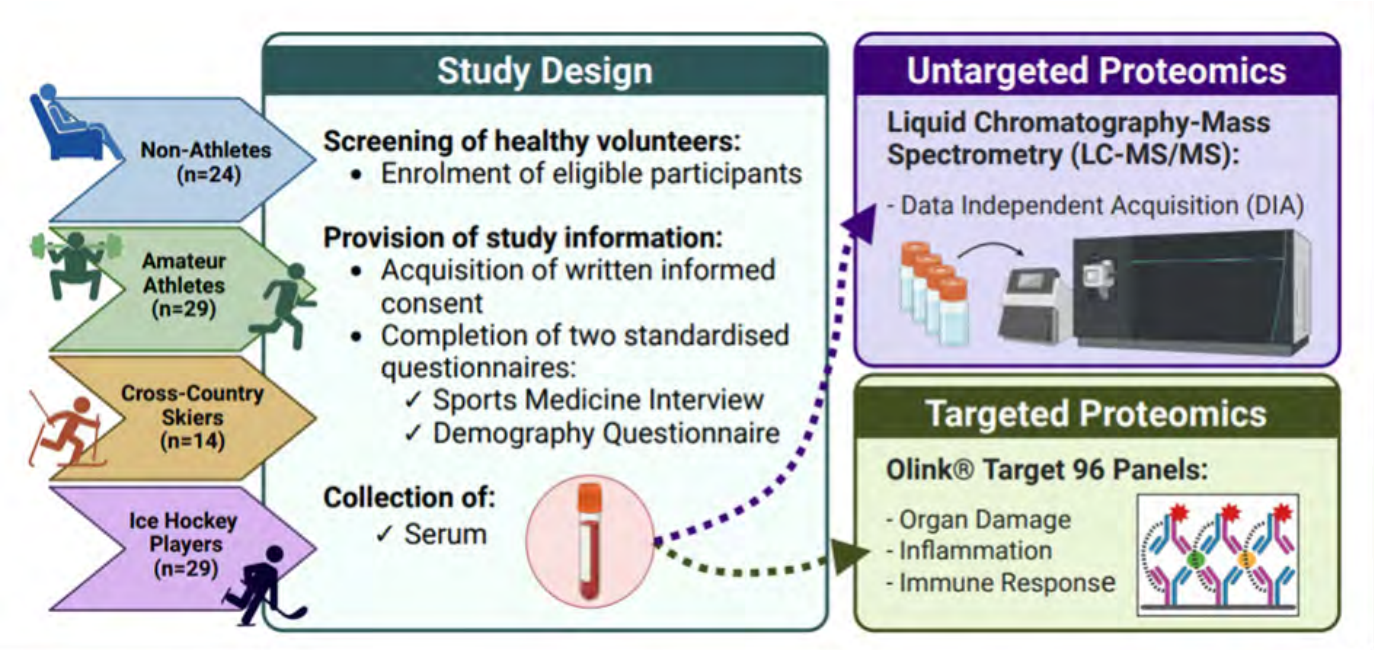


Figure 3: Fig. S1 in the Supporting Information with an overview of the study design. Eligible healthy volunteers were enrolled following screening, informed consent, and completion of two standardised questionnaires, before blood collection for downstream targeted and untargeted proteomics analyses (Barletta et al., submitted).

Davos, May 2026

Dr. Claudio Rhyner, PhD

Topic: Veterinary allergology

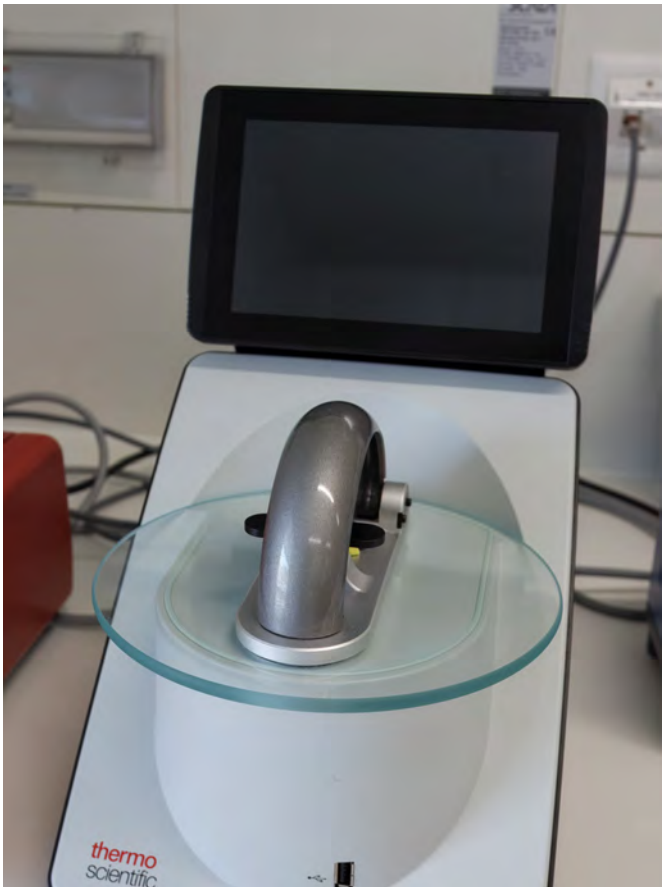
Equine asthma (EA) is a common disease of adult horses with chronic respiratory pathology and common neutrophilic airway inflammation. It presents with hyperreactivity to hay dust components such as molds, and underlying dysregulated T cell responses have been suggested. Thus far, T cells have been analysed in EA with conflicting results and the antigen reactivity of T cells has not been demonstrated. Serological and epidemiological data point to the relevance of Aspergillus fumigatus as an antigen source in EA. Serum and BALF from healthy and asthmatic horses were compared. Ig isotype (IgG1, IgG3/5, IgG4/7, IgG6, IgA and IgE) binding to nine A. fumigatus antigens was compared by ELISA. Total Ig isotype contents were determined by bead-based assays. We delivered purified, recombinant allergens.

Topic: Atopic dermatitis

There are conflicting data on a potential association between atopic dermatitis (AD) and cardiovascular diseases, as a potential comorbidity. In a study with 677 AD patients and 79 nonatopic controls from an observational multicenter case-control study (ProRaD: Prospective longitudinal study investigating the remission phase in patients with atopic dermatitis and other allergy-associated diseases). AD severity and atopic, metabolic, and cardiovascular conditions as well as risk factors were assessed. Serum samples were analyzed with targeted proteomics (cardiometabolics panel, Olink). No overall association between AD and CVD could be proven. However, AD patients without atopic comorbidities (pure AD) showed a significantly higher CVD prevalence than AD patients with atopic comorbidities (ADAC) (28.2% [37/131] vs. 14.7% [80/546], $p < 0.001$). Yet, this association could not be confirmed when controlling for cardiovascular risk factors.

Deciphering the Connection Between Atopic Dermatitis and Cardiovascular Diseases: Analysis of Clinical Associations and Cardiometabolic Proteins.
Fehr D, Huynh-Tran VH, Maintz L, Niederseer D, Ameri M, Dreher A, Study Group CC, Akdis CA, Lauener R, Rhyner C, Traidl-Hoffmann C, Schmid-Grendelmeier P, Bieber T, Brüggen MC.Allergy. 2025 Aug;80(8):2187-2200. doi: 10.1111/all.16588. Epub 2025 May 19.PMID: 40386898

Davos, May 2026



Immune Metabolism

PD Dr. Milena Sokolowska, MD

Changes in environmental conditions and lifestyle choices have contributed to an increase in allergies, respiratory viral infections, and bacterial illnesses. The COVID-19 pandemic, along with its aftermath in the form of long COVID, has starkly highlighted this trend. The underlying reasons why certain substances trigger allergic reactions in some individuals while others remain unaffected remain elusive. Similarly, the factors determining susceptibility to viral infections, the development of severe respiratory illnesses, or long-lasting consequences such as long COVID, remain poorly understood. Various factors can contribute to this susceptibility, including inadequate early-life microbiome development, recurrent viral infections, exposure to environmental pollutants, imbalanced diets, and metabolic disorders like obesity. In recent years, concerted efforts have focused on unraveling the susceptibility to viral infections in individuals with asthma and allergies, as well as the role of allergens and allergic inflammation in these complex dynamics.

Our research group has focused on how the above-mentioned factors can disrupt the intricate communication between airway epithelial cells and CD4+T cells, impacting cellular responses at a metabolic and molecular level. Immune cells must undergo numerous energetically demanding processes to react to external stimuli such as allergens, viruses, or bacteria.

These processes involve gene expression alterations, protein translation, lipid synthesis, activation of signaling pathways, cytoskeletal modifications, and the production of cytokines, lipid mediators, as well as cell proliferation or migration. Cellular energy metabolism in the form of glycolysis, the tricarboxylic acid cycle, oxidative phosphorylation, and beta-oxidation needs to sustain the changing needs of immune cells during homeostasis and inflammation or infection in the processes called metabolic reprogramming. To understand the complex interplay between immune and metabolic reprogramming at mucosal barriers in the pathogenesis of allergic, respiratory, skin and autoimmune diseases, our research group employs several advanced techniques. We analyze patients' blood, lung, and skin samples using single-cell and bulk sequencing, spatial transcriptomics, proteomics, and metabolomics, coupled with comprehensive bioinformatic approaches. We also combine these with CRISPR gene editing, multi-color flow cytometry and sorting, confocal microscopy, and live-cell metabolic assays such as single-cell energy metabolism profiling to understand mechanisms of observed pathologic phenomena. Our overarching goal is to decipher immune and metabolic crosstalk on a single cell level and multi-omic level, identify new biomarkers, and uncover and test potential targets for prevention and treatment across various respiratory viral diseases, microbial dysbiosis, allergy, immune tolerance, and asthma phenotypes.

1. **Immunometabolism in viral infections, immune tolerance, allergy and asthma**

L-Phenylalanine is a metabolic checkpoint of human Th2 cells
Kulkarni AJ, Rodriguez-Coira J, Stocker N, Radzikowska U, García-Cívico AJ, Delgado Dolset MI, Contreras N, Jardón Parages I, Saiz Sanchez V, Serrano P, Izquierdo E, Gomez-Casado C, Sanchez-Solares J, Pablo-Torres C, Obeso D, Moreno-Aguilar C, Espinazo ML, Eljaszewicz A, Koch J, Baerenfaller K, Heider A, Tan G, Zhakparov D, Escribese MM, Ruiz-Leon B, Akdis CA, Argüello RJ, Barber D, Villaseñor A, Sokolowska M. Cell Rep Med. 2025 Dec 16;6(12):102466. doi: 10.1016/j.xcrm.2025.102466.

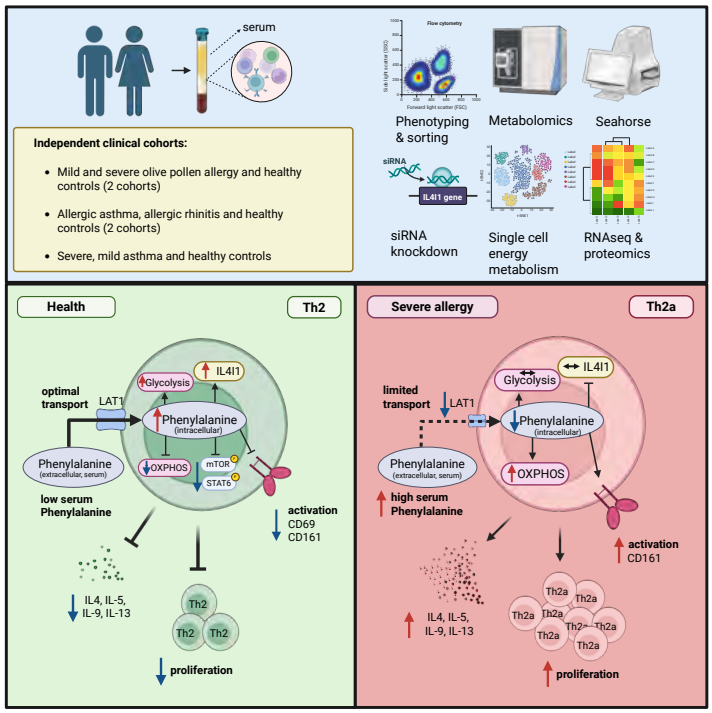


Figure 1.
*Graphical summary of Kulkarni A**, Rodriguez-Coira J**, et al and Villaseñor A*, Sokolowska M*. Cell Rep Med. 2025 Dec 16;6(12):102466. **equal contribution. *equal senior authorship.*

After the primary response, circulating memory CD4+T effector and T regulatory (Treg) cells regulate recall responses, typically impaired in allergy. We discovered distinct metabolomes of these cells in humans, differentially enriched in phenylalanine-related metabolites. Energy metabolism assessment in in vitro and ex vivo single-cell analyses revealed that increased intracellular L-phenylalanine boosts glycolysis while limiting oxidative phosphorylation (OXPHOS) in CD4+T, memory CD4+T, and Th2 cells, but not in Th1, Th17, or Treg cells. L-phenylalanine also restrains proliferation of memory CD4+T, Th2, and Th17 cells in an IL4I1-dependent manner and limits Th2 differentiation via inhibition of STAT6 and mechanistic target of rapamycin (mTOR) signaling. RNA sequencing, metabolomics, flow cytometry, and proteomics, validated both in vitro and across patient cohorts, revealed impaired LAT1-dependent transport of L-phenylalanine into Th2 cells in allergy, with increased intracellular processing accompanied by expansion of pathogenic Th2 cells. Thus, our study identifies L-phenylalanine as a checkpoint in Th2 cell development, energy metabolism, and function (Figure 1).

Dynamic Regulation of Collagens, Proteases, Their Inhibitors, and Cell Death in Experimental Asthma in Mice

Tynecka M, Tarasik A, Hanczaruk B, Janucik A, Czolpinski K, Walewska A, Zbikowski A, Zeller A, Kulczynska-Przybik A, Niemira M, Reszec-Gielazyn J, Mroczko B, Kretowski A, Akdis CA, Sokolowska M, Moniuszko M, Karaaslan C, Eljaszewicz A. Allergy. 2026 Jan;81(1):170-184. doi: 10.1111/all.70126.

Asthma is a complex airway disorder driven by diverse immunological pathways. While type 2 (T2)-mediated inflammation is well characterized, the mechanisms underlying T2-low (also referred to as mixed inflammatory phenotype) or non-T2-mediated asthma, often associated with Th1/Th17-driven immune responses and high pulmonary neutrophilic inflammation, remain poorly understood. This study investigates airway remodeling in acute and chronic experimental asthma models induced by house dust mite extract to elucidate inflammatory and structural changes. Two acute T2-low models demonstrated pronounced airway inflammation characterized by goblet cell hyperplasia, collagen deposition, and significant upregulation of extracellular matrix remodeling and fibroblast activation. In contrast, the chronic Th17-mediated model exhibited reduced ECM deposition, increased matrix metalloproteinase (MMP) activity, and a distinct molecular signature dominated by IL-17A-driven pathways, indicative of ECM degradation and structural instability. Furthermore, the acute models showed immune cell apoptosis as the predominant cell death mechanism. In contrast, the chronic inflammation model was marked by necroptosis localized to structural lung cells, contributing to persistent inflammation and remodeling. Our findings provide new insights into the distinct immunopathological mechanisms underlying airway remodeling and fibrosis in T2-low and Th17-mediated asthma phenotypes. The study emphasizes the need for advanced preclinical models and targeted therapeutic strategies to address ECM dysregulation and chronic inflammation to progress in airway remodeling treatment in asthma.

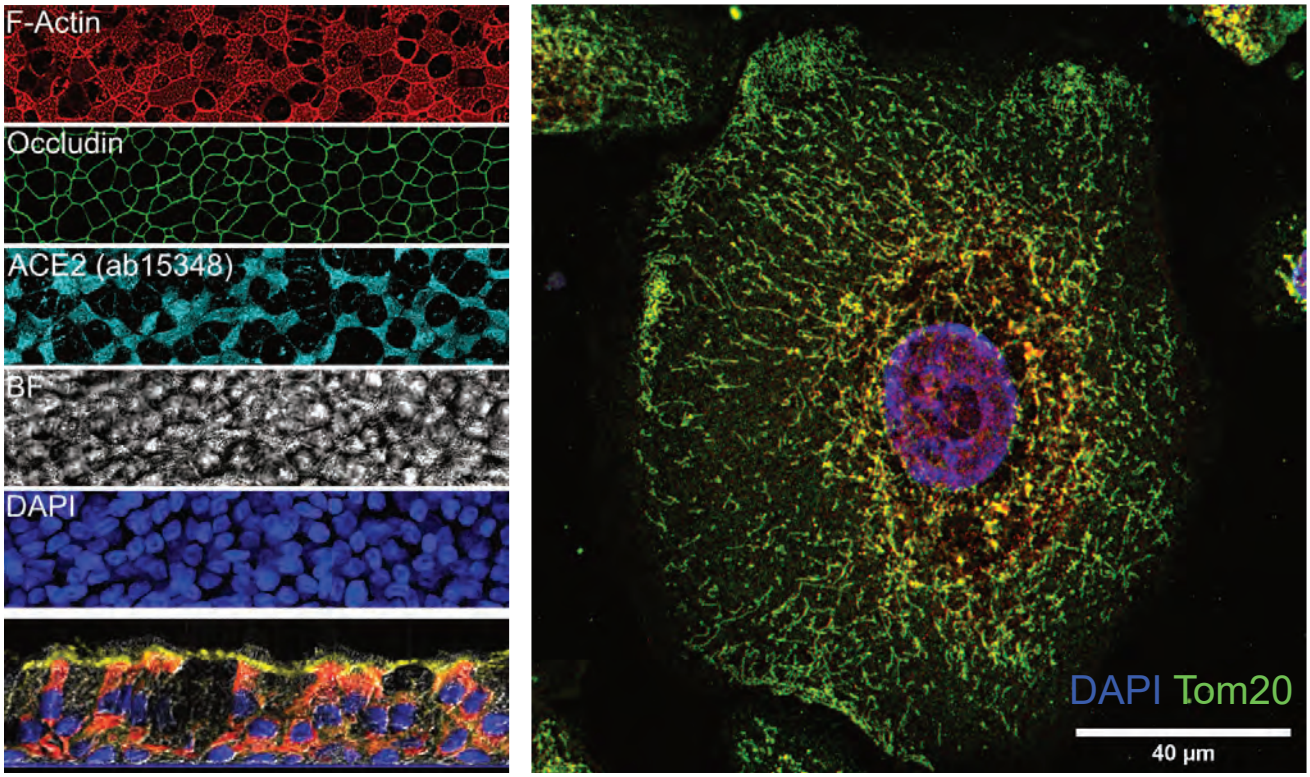


Figure 2. Mitochondrial stress in human primary bronchial epithelial cells after infection with respiratory viruses.
A) Human bronchial epithelial cells in air-liquid interphase (ALI) from above stained with F-actin for cilia, occludin for tight junctions, ACE2, for vSARS-CoV-2 viral entry, bright field (BF) and nuclei (DAPI) or combined in transversal cut. B) Mitochondria in primary human bronchial epithelial cells stained with Tom20 (green) and DAPI (blue). (Radzikowska U, Jardón Parages I* et al and Sokolowska M. *equally contributed. UNPUBLISHED).*

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Immunometabolic reprogramming of bronchial epithelium in asthma and during viral infection.
Radzikowska U, Jardón Parages I *et al and Sokolowska M. * equally contributed. In preparation*

Rhinovirus (RV) exacerbates asthma in both children and adults, yet little is known about the immunometabolic changes occurring in airway epithelium in response to RV infection. Our study aims to elucidate abnormalities in mitochondria, oxidative phosphorylation (OXPHOS), beta-oxidation, and glycolysis in asthmatic airway epithelium, both at baseline and post-RV infection. We employed a multidimensional approach encompassing RNA-seq, qPCR, untargeted and targeted proteomics (LC-MS and OLINK), immunoassays, metabolomics, microscopy, and Seahorse and single cell energy metabolic profiling (SCENITH) to examine human primary bronchial epithelial cells (HBECs), as well as bronchial biopsies from healthy controls and asthma patients, including experimental in vivo infection with RV in humans (Figure 2). Our results demonstrate that bronchial epithelium in asthma, both in vitro and in vivo, exhibits defective OXPHOS energy generation at baseline and after RV infection, coupled with an increased reliance on glycolysis. This metabolic shift is functionally linked to abnormal mitochondrial dynamics and function, as well as enhanced early RIG-I inflammasome activation, culminating in an inefficient antiviral response and unresolved airway inflammation. Transcriptomic analyses further revealed dysregulation of OXPHOS and glycolysis genes in asthma post-infection, potentially contributing to impaired viral clearance and ATP regulation. Moreover, proteomic assessments highlighted alterations in metabolism-related proteins, particularly those involved in OXPHOS, emphasizing the significance of epithelial metabolic rewiring in asthma exacerbations induced by RV. Our findings underscore the importance of epithelial metabolic rewiring in asthma exacerbations induced by RV, suggesting it as a potential target for therapeutic interventions aiming to prevent and treat viral exacerbations of asthma.

Interleukin-13 and interleukin-22 differentially regulate glycolysis in keratinocytes of patients with atopic dermatitis.
Mitamura Y, Duphey S* et al and Sokolowska M. * equally contributed. In preparation.*

Atopic dermatitis (AD) is a chronic skin disease. Interleukin-13 (IL-13) and interleukin-22 (IL-22) play the central role in the pathogenesis of AD. However, the influence of these cytokines on the keratinocytes metabolism and metabolic control of epithelial barrier remains unknown. Recently, an enhanced glycolysis pathway has been reported in AD. We aim to investigate the roles of IL-13 and IL-22 on the metabolic change in keratinocytes and their subsequent influence on barrier function. Data of bulk RNA sequencing and spatial RNA sequencing of lesional and non-lesional skin of AD patients in comparison to healthy skin from our previous cohort were analysed. In addition, we performed bulk RNA sequencing of IL-13-stimulated-reconstructed human skin (Episkin®). Next, we analysed real time glycolysis utilization by proliferating and differentiating keratinocytes in response to IL-13 and IL-22 in the seahorse glycolysis stress test. Finally, we performed qPCR and confocal microscopy experiments in air-liquid interphase cultured keratinocytes to investigate gene and protein expression of barrier- and glycolysis-related molecules. The expression of glycolysis-related genes was significantly upregulated especially in the epidermis in the lesional skin of AD patients and in the IL-13-treated Episkin, whereas the expression of several barrier molecules was downregulated. In the seahorse experiments, IL-13 significantly increased, whereas IL-22 decreased the early glycolytic capacity of basal proliferating and differentiating keratinocytes. We further demonstrated that functional blocking of glycolysis by 2-deoxy-d-glucose (2-DG) influenced the expression of some of the keratinocyte-specific molecules. Our data suggest that IL-13 and IL-22 may take the balance to regulate the glycolytic capacity of keratinocytes, which might further affect the skin proliferation, differentiation, and barrier regulation in AD.

Single-cell metabolic profiling of airway epithelium reveals compensatory metabolism during viral infection.
Jardón Parages I et al and Sokolowska M. In preparation.

The bronchial airway epithelium is the primary site of respiratory viral infection and a key initiator of immune responses, yet its metabolic heterogeneity remains poorly resolved. Existing approaches lack the resolution to interrogate metabolism across epithelial subsets, and single-cell methods such as SCENITH (single-cell energetic metabolism by profiling translation inhibition) have not been adapted to structurally complex tissues. Here, we combine an expanded flow cytometry panel with an adapted SCENITH workflow to enable single-cell metabolic profiling of differentiated human airway epithelium cultured under air-liquid interface conditions. This approach resolves major epithelial subsets and reveals high glycolytic contribution to translation, variable mitochondrial involvement and donor-dependent metabolic adaptability, including cases where mitochondrial inhibition increases translation (Figure 3).

WIRM, Group photo Immune Metabolism

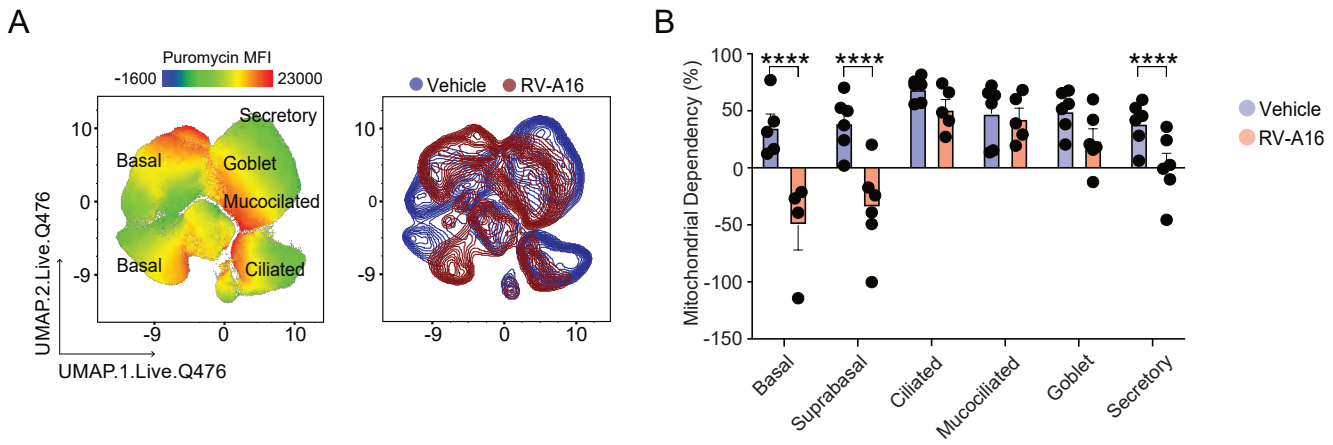


Figure 3. Rhinovirus A16 infection differentially rewires mitochondrial dependency in epithelial cell subsets.
A, B) Single Cell Energetic Metabolism by Profiling Translation Inhibition (SCENITH) revealing shutting down mitochondrial oxidative phosphorylation in several subsets of airway epithelial cells after infection with Rhinovirus A16 (Jardón Parages I et al and Sokolowska M. *equally contributed. UNPUBLISHED).*

2. Immunology of viral infections, asthma exacerbations and their long-term consequences

Barrier dysfunction in nasal epithelium contributes to persistent inflammation in long COVID

Baalbaki N, van Egmond D, Jaeger P, Cornelissen MEB, Verbeek ST, Sokolowska M, van Drunen CM, Maitland-van der Zee AH, Golebski K; P4O2 consortium. J Allergy Clin Immunol. 2025 Oct 8:S0091-6749(25)01022-X. doi: 10.1016/j.jaci.2025.09.024.

Long COVID (LC) is characterized by persistent symptoms associated with chronic inflammation and immune dysregulation, but the local tissue mechanisms driving these processes remain poorly understood. Given that the nasal epithelium is the primary entry and infection site for SARS-CoV-2, we aimed to investigate its role in LC and its potential contribution to systemic immune activation. We analyzed nasal epithelial samples and peripheral blood from participants in the Precision Medicine for More Oxygen (P4O2) COVID-19 cohort, post-COVID-19 individuals, and healthy controls. We assessed epithelial barrier integrity, evaluated wound healing, and explored cytokine profiles. Transcriptomic analysis was performed via RNA sequencing. Blood innate lymphoid cells (ILCs) were phenotyped by flow cytometry and stimulated in vitro for functional assays. Among a subgroup of LC patients, nasal epithelial cells showed impaired barrier function, reduced expression of ZO-1 and occludin and exaggerated sensitivity to viral triggers. Despite faster wound closure, the epithelial repair was reduced. The LC nasal epithelium exhibited increased cytokine production, including IL-1β and transcriptomic signatures of inflammation, including upregulation of interferon pathways. Furthermore, we found that TFs ATF3 and EGR1 were downregulated in LC. Elevated IL-1β levels in nasal epithelium promoted ILC activation and plasticity toward IFN-γ-producing ILCs in blood. While multiple organ systems are implicated in LC, our findings identified nasal epithelial dysfunction in a subgroup of LC patients and chronic activation as potential contributors to systemic immune dysregulation. The IL-1β-IFN-γ axis represents a novel targetable pathway that may support precision therapies for LC.

Cannabinoid WIN55,212-2 restores bronchial epithelium by regulating oxidative stress and STAT6 phosphorylation

Pérez-Diego M, Angelina A, Pat Y, Maldonado A, Sevilla-Ortega C, Martín-Cruz L, Yazici D, Rückert B, Sokolowska M, Martín-Fontecha M, Akdis M, Akdis CA, Palomares O. J Allergy Clin Immunol. 2025 May 16:S0091-6749(25)00551-2. DOI: 10.1016/j.jaci.2025.05.002

Viral infections and type 2 immune responses perpetuate airway epithelial barrier dysfunction and inflammation, leading to the development and progression of asthma. The synthetic cannabinoid WIN55,212-2 displays anti-inflammatory properties by acting on different immune system cells. We sought to investigate the capacity of WIN55,212-2 to restore bronchial epithelial barrier function in asthma in the context of viral infections or type 2-driven inflammation. Air-liquid interface cultures of human bronchial epithelial cells and human bronchial epithelial spheroids were generated to assess the capacity of WIN55,212-2 to restore airway epithelial barrier damage induced by human rhinovirus A16 (RV-A16) infection or type 2 inflammation. RT-PCR, cytokine quantification, permeability assays, metabolic studies, flow cytometry, and Western blot techniques were employed to assess the effects of WIN55,212-2 on the airway epithelium. The in vivo relevance of our findings was evaluated in a murine model of IL-13-induced airway inflammation. Prophylactic and therapeutic administration of WIN55,212-2 accelerated the recovery from RV-A16-induced bronchial epithelial barrier damage. WIN55,212-2 inhibited the acquisition of IL-13-induced type 2 asthma features in air-liquid interface cultures, self-assembled bronchial epithelial spheroids, and in vivo asthma model of airway inflammation and epithelial dysfunction. Mechanistically,

WIN55,212-2 impaired IL-13-induced oxidative stress in epithelial cells, restoring the activity of protein tyrosine phosphatases, which in turn inhibited pSTAT6-mediated signaling pathways and asthma features. The cannabinoid WIN55,212-2 displays airway epithelial barrier protective effects during RV-A16 infection or type 2 inflammation by mechanisms associated with the modulation of oxidative metabolism and pSTAT6-mediated signaling.

STING-dependent induction of neutrophilic asthma exacerbation in response to house dust mite

Messaoud-Nacer Y, Culerier E, Rose S, Maillet I, Boussad R, Veront C, Savigny F, Malissen B, Radzikowska U, Sokolowska M, da Silva GVL, Edwards MR, Jackson DJ, Johnston SL, Ryffel B, Quesniaux VF, Togbe D. Allergy. 2025 Mar;80(3):715-737. doi: 10.1111/all.16369.

Severe refractory, neutrophilic asthma remains an unsolved clinical problem. STING agonists induce a neutrophilic response in the airways, suggesting that STING activation may contribute to the triggering of neutrophilic exacerbations. We aim to determine whether STING-induced neutrophilic lung inflammation mimics severe asthma. We developed new models of neutrophilic lung inflammation induced by house dust mite (HDM) plus STING agonists diamidobenzimidazole (diABZI) or cGAMP in wild-type, and conditional-STING-deficient mice. We measured DNA damage, cell death, NETs, cGAS/STING pathway activation by immunoblots, N1/N2 balance by flow cytometry, lung function by plethysmography, and Th1/Th2 cytokines by multiplex. We evaluated diABZI effects on human airway epithelial cells from healthy or patients with asthma and validated the results by transcriptomic analyses of rhinovirus infected healthy controls vs patients with asthma. DiABZI administration during HDM challenge increased airway hyperresponsiveness, neutrophil recruitment with prominent NOS2+ARG1- type 1 neutrophils, protein extravasation, cell death by PANoptosis, NETs formation, extracellular dsDNA release, DNA sensors activation, IFNγ, IL-6 and CXCL10 release. Functionally, STING agonists exacerbated airway hyperresponsiveness. DiABZI caused DNA and epithelial barrier damage, STING pathway activation in human airway epithelial cells exposed to HDM, in line with DNA-sensing and PANoptosis pathways upregulation and tight-junction downregulation induced by rhinovirus challenge in patients with asthma. Our study identifies that triggering STING in the context of asthma induces cell death by PANoptosis, fueling the flame of inflammation through a mixed Th1/Th2 immune response recapitulating the features of severe asthma with a prognostic signature of type 1 neutrophils.

3. Microbiome and nutrition in prevention of allergic diseases

Scoping review of the association between complementary feeding, growth, and immune health in infants and toddlers-An EAACI guideline paper

Venter C, Alonso-Coello P, Beltran J, Bracchiglione J, Fernandez-Saenz K, Riera P, Solà I, Arasi S, Berni Canani R, Fleischer D, Eguíluz-Gracia I, Meyer R, Roberts G, Roth-Walter F, Santos AF, Skypala I, Smith PK, Sokolowska M, Torres MJ, Vassilopoulou E, Walter J, O'Mahony L. Pediatr Allergy Immunol. 2025 Aug;36(8): e70145. doi: 10.1111/pai.70145

The European Academy of Allergy and Clinical Immunology (EAACI) is currently developing guidelines on immunomodulation and nutrition. To inform these recommendations, a scoping review will be conducted to synthesize and map the available empirical evidence on how complementary feeding affects immune health in infants and toddlers to explore the association between complementary feeding during the first year of life and immune health outcomes in children up to 3 years of age. The scoping review will be conducted using the Preferred Reporting Items for Systematic Reviews and Meta-analysis Scoping Review Extension for scoping reviews (PRISMA-ScR) guidelines. We have developed inclusion criteria using the PCC (Population, Concept and Context) format: the population will include healthy infants; the concept refers to specific factors related to introduction of complementary feeding; and the context will focus on studies conducted in settings representative of everyday nutrition and feeding practices for infants and toddlers. Evidence sources include systematic reviews. We will search in MEDLINE and Epistemonikos. Data will be extracted using a standardized form developed in REDCap, and the findings will be synthesized narratively. Only published literature will be included in this systematic review. Therefore, no ethical approval is required. The final review paper will be published in a peer-reviewed journal. OSF registration DOI: 10.17605/OSF.IO/T5F8.

Microbiota-Derived Tryptophan Metabolites Shape TH2 Lymphocyte Responses Via the Aryl Hydrocarbon Receptor and Metabolic Reprogramming

Lu Yao, Tanya Guevara Avella&, Abhijeet J Kulkarni&, Marlinde Waardenburg&, Maria Isabel Delgado-Dolset&, Avril Walters, Kristoff Nieves, Hannah Devotta, Songhui Kim, Emer Shannon, Nonhlanhla Lunjani, Douwe van Sinderen, Jens Walter, Milena Sokolowska, Liam O'Mahony*. &equally contributed. *equal senior authorship. In preparation*

Allergic diseases have been consistently linked with changes in gut microbiota composition and metabolism, but the direct mechanisms linking microbial-derived metabolites with the modulation of T helper 2 (TH2) lymphocyte differentiation are still poorly understood. Colonisation of gnotobiotic animals with Bifidobacterium longum and Clostridium sporogenes resulted in high levels of the tryptophan-derived indole-3-lactic acid (ILA), indole-3-acrylic acid (IA), and indole-3-propionic acid (IPA), which correlated with suppressed TH2 responses. Indoxyl-3-sulfate (I3S) and IA reduced IL-4, IL-5 and IL-13 secretion from human TH2 polarised lymphocytes in an aryl-hydrocarbon receptor (AHR)-dependent manner. However, IPA suppression of IL-4 expression was AHR independent. RNA sequencing of TH2 lymphocytes suggested potent modulation of oxidative phosphorylation (OXPHOS) in indole-exposed TH2 lymphocytes. This was accompanied by reduced reactive oxygen species (ROS), lipid peroxidation and growth differentiation factor-15 (GDF-15) secretion. IA and IPA induced broader metabolic rewiring by decreasing fatty acid and amino acid utilisation and reducing mitochondrial oxidative phosphorylation. In fully differentiated TH2 cells, indoles inhibited glycolysis, OXPHOS and cytokine expression in a STAT6 independent manner. Collectively, these findings demonstrate that microbial tryptophan metabolism may mediate one

of the mechanisms linking allergy risk with changes in the microbiome. In addition, we have identified novel associations between mitochondrial metabolic programs and TH2 lymphocyte polarisation that, in particular, are impacted by the microbial Stickland pathway derived metabolites IA and IPA.

4. Multi-omics approaches and systems medicine in allergy and immunology

The Use of Omics Sciences in Allergic Diseases From Bench to Bedside: An EAACI Position Paper

Tontini C, Radzikowska U, Aranda CJ, Tynecka M, Dolset MID, Eljaszewicz A, Cornejo-García JA, Villaseñor A, Ariza A, Contreras N, López-Rodríguez JC, Karaguzel D, Lozano-Ojalvo D, Castagnoli R, Eguiluz-Gracia I, Sokolowska M, Mayorga C, Hilger C, Mortz CG, Pfaar O, Barber D, Escribese MM, Karaaslan C. Allergy. 2025 Sep;80(9):2442-2483. doi: 10.1111/all.16638.

Omics have revolutionized our understanding of allergic diseases, with increasingly more studies adopting techniques like genomics, proteomics, and metabolomics over the years. Integrating high-dimensional omics data with clinical features can enable more precise disease classification and biomarker identification, leading to improved disease management and the development of novel therapies. However, translating these discoveries into clinical practice remains a challenge. In this European Academy of Allergy and Clinical Immunology (EAACI) Position Paper, the Task Force (TF) "The Use of Omics Sciences in Asthma and Allergy Clinical Practice" performed a broad review of recent papers where omics technologies were used in the context of clinical studies and provided a summary of relevant findings in the field of asthma, atopic dermatitis, allergic rhinitis, food allergy and drug hypersensitivity reactions. Particular attention was dedicated to studies investigating overlapping diseases/co-morbidities, revealing unique and shared signatures that could guide future therapeutic and research efforts. Furthermore, current hurdles in translating findings from bench to bedside were explained, and possible solutions were offered. With this position paper, the TF aims to assist researchers and clinicians by providing an overview of the investigations performed to date, highlighting gaps in research, limitations, and avenues for further exploration in clinical practice.

Multiomics approach to evaluating personalized biomarkers of allergen immunotherapy

Shamji MH, Fulton WT, Animashaun I, Palmer E, Baerenfaller K, Sokolowska M, Barber D, Huffaker M, Baloh C, Pfaar O, Ollert M, Klimek L, Rabin RL, Tripathi A, Togias A, Vieths S, Shreffler WG, Layhadi JA. J Allergy Clin Immunol. 2025 Sep;156(3):523-534. doi: 10.1016/j.jaci.2025.06.036.

Recent advancements in genomics and "omic" technologies have ushered in a transformative era referred to as personalized or precision medicine. This innovative approach considers the unique genetic profiles of individuals, along with a range of variability factors, to devise tailored disease treatments and prevention strategies that cater to the distinct needs of each patient. Although the terms personalized medicine and precision medicine are frequently utilized interchangeably, it is essential to delineate the subtle distinctions between them. Personalized medicine concentrates on bespoke treatments and prevention strategies that are meticulously tailored for each individual. Conversely, precision medicine utilizes advanced gene sequencing and comprehensive data analytics to formulate specific treatments and prevention strategies for defined groups of individuals based on genetic, environmental, and lifestyle factors rather than focusing exclusively on individual patients. Therefore, precision medicine constitutes an extension of traditional personalized care, improving the accuracy of diagnosis, prognosis, and therapy estimations for each patient through the application of sophisticated molecular diagnostics and advanced imaging techniques enabled by recent technological innovations. The shift from personalized to precision medicine has gained considerable traction, particularly in the wake of the 2015 US Precision Medicine Initiative, which has further stimulated advancements in this domain. This review will explore multiomics approaches that have facilitated the evaluation of personalized biomarkers associated with allergen immunotherapy, particularly in the treatment of allergic diseases. By leveraging these innovative methodologies, we aspire to cultivate more effective and individualized patient care, ultimately enhancing health outcomes for individuals with allergies.

5. Novel avenues for biomarkers and precision medicine in allergy, asthma and clinical immunology

Molecular Signatures and Functional Pathways of Human Monocytes and Macrophages in Allergy: An EAACI AllergoOncology Scoping Review

Bianchini R, Escolar-Peña A, Pick V, Poli A, Adams R, Basilio J, Cari L, Chauhan J, Chivato T, Vecillas LL, Delgado-Dolset MI, Escribese MM, Grandits M, Bax HJ, Martín-Antoniano IA, Martín-Cruz L, Mayerhofer H, Michelucci A, Nocentini G, Osborn G, Pablo-Torres C, Palomares O, Pascal M, Radzikowska U, Rohr-Udilova N, Sokolowska M, Bergmann C, Jensen-Jarolim E, Karagiannis SN, Rebollido-Rios R, Izquierdo E. Allergy. 2025 Oct;80(10):2710-2725. doi: 10.1111/all.16672.

AllergoOncology explores the intersection of allergic diseases and cancer, focusing on shared immune mechanisms. While monocytes and macrophages are extensively studied in cancer, their roles in allergic diseases remain underexplored. To address this gap, we conducted a scoping review to systematically characterize the molecular landscape and related pathways of human monocytes and macrophages in allergy. An automated search of PubMed and Web of Science databases retrieved 4668 unique articles, which were manually curated based on predefined inclusion and exclusion criteria, yielding 138 eligible studies. From these, we identified 451 molecules associated with monocyte and macrophage responses across allergic disorders. Data analyses revealed a research bias towards blood-derived monocytes, underrepresentation of tissue-resident macrophages, and limited inclusion of miRNAs. Semantic similarity and pathway enrichment analyses highlighted a common molecular signature across major allergic disorders, with consistent enrichment in interleukin signaling and immune activati-

on pathways. To enhance reproducibility and translational utility for researchers and clinicians, we developed ALO•HA, a web application for interactive data exploration. This overview of monocyte and macrophage molecular responses in human allergy underscores the need for integrative, human-focused approaches to better define their roles, and to guide future therapeutic strategies in allergic diseases and at the interface with oncology.

Endotypes in Immune Mediated Drug Reactions: Present and Future of Relevant Biomarkers. An EAACI Task Force Report.
Mayorga C, Fernandez-Santamaria R, Çelik GE, Labella M, Murdaca G, Sokolowska M, Naisbitt D, Sabato V. Allergy. 2025 Jul;80(7):1831-1847. doi: 10.1111/all.16576.

Drug-induced immune reactions are an important burden for patients and health systems. They can be classified into immediate-drug hypersensitivity reactions (IDHRs) and delayed-DHRs (DDHRs) based on their phenotype. Drugs do not always behave as allergens and need to bind to proteins, forming adducts. Therefore, IDHRs can be classified as antigenic (IgE, and IgG mediated) and nonantigenic immune responses (complement activation-[CARPA], mas-related G-protein coupled receptor member X2 [MRGPRX2], cyclooxygenase [COX]-1 and cytokine release reactions [CRRs]). DDHRs are even more complex due to the different cell subsets and mechanisms involved, showing both antigenic and nonantigenic immune responses too. Since different endotypes result in similar phenotypes, the establishment of specific biomarkers is essential for an accurate diagnosis, with important relevance for the management of patients, as well as for risk stratification. The biomarkers of clinical utility are skin tests, specific IgE (sIgE), tryptase, and some HLA-DR genotyping. The diagnostic performance depends on the responsible drug. This review highlights that, unfortunately, most biomarkers have not gone beyond analytic or clinical validity. It is therefore important to set up multicentre translational studies to advance the validation process towards reaching a clinical utility phase.

Novel candidate biomarkers of the efficacy of allergen immunotherapy. Van Elst D*, Agache I*, Ram I et al and Sokolowska M. * equally contributed. In preparation.

Allergen immunotherapy (AIT) is the only curative treatment for patients suffering from allergic diseases such as allergic rhinitis (AR) and allergic asthma by inducing allergen-specific immune tolerance. To find potential biomarkers of efficacy of AIT we implemented an untargeted proteomics comparison of serum obtained 2 years after the start of AIT against house dust mite, grass pollen or cat antigens, from adults and children suffering from AR and allergic asthma. The control group included age- and gender-matched healthy controls. Serum proteomics data, obtained by mass spectrometry in a data-independent mode were compared between non-responders (NR) and responders to the therapy (R), and between NR and controls. Biomarker assessment and network association analyses were performed to investigate activated pathways and protein interactions. We detected 166 serum proteins in adults and 223 in children. In adults, six proteins were differentially expressed in NR as compared to R. No differentially expressed proteins were detected between NR and R in children. Western blot (WB) analysis of some of these candidate proteins validated these findings. When comparing enriched process networks in NR versus R, several inflammation, metabolic and the immune response related processes were significantly enriched. Interestingly, we found that distinctly identified biomarker candidates contribute to these processes. Identified potential biomarkers in the present study could provide an important step in understanding the long-term effects of AIT and in implementing a personalised medicine approach allowing selection of responders to AIT.

Update on Non-Biological and RNA-Based Therapeutics in Chronic Inflammatory Diseases: Precision Medicine Through Small Molecules: An EAACI Position Paper
Roth-Walter F, Adcock IM, Benito-Villalvilla C, Bianchini R, Bjermer L, Caramori G, Cari L, Chung KF, Diamant Z, Eguiluz-Gracia I, Knol EF, Jesenak M, Levi-Schaffer F, Nocentini G, O'Mahony L, Palomares O, Redegeld F, Sokolowska M, Van Esch B, Zurlo M, Stellato C. Allergy. 2026 Feb 7. doi: 10.1111/all.70235.

In the last decades, critical advancements in research technology and knowledge on disease mechanisms steered therapeutic approaches for chronic inflammatory diseases towards unprecedented target specificity. For allergic and chronic lung diseases, biologic drugs pioneered this goal, acquiring on the way-through the clinical use of monoclonal antibodies-a deeper understanding of how inflammatory and immune pathways are configured in disease-specific patterns. In this biomarker-driven approach, synthetic small molecule drugs (SMDs) were perceived as lagging behind in innovation for their relative lack of specificity. This was, however, mostly due to a shift in focus towards biologics rather than true obsolescence of SMDs. In the same timeframe, in fact, advances in structural biology and medicinal chemistry, bioinformatics and artificial intelligence held steadily SMDs' innovation and relevance. The use of kinase inhibitors, well established in the treatment of cancer and rheumatological diseases, is now approved for some allergic skin diseases and is approaching asthma and COPD with several clinical trials; moreover, new therapeutics targeting mast cell receptors and molecules involved in innate immunity are entering preclinical and clinical testing. Alongside, the portfolio of biologics is harboring the expansion of RNA therapeutics, which gained global recognition during the COVID-19 pandemic due to RNA vaccines. Different types of RNA therapeutics, including those based on different non-coding RNAs, are advancing to agency approval and market, thanks to improvements in molecule stability and delivery systems. In summary, the evidence presented in this position paper illustrates that precision medicine is becoming a goal shared between synthetic SMDs and biologics, both protein/antibody-based and RNA therapeutics. We review the current state, unmet needs and opportunities within this evolving landscape, highlighting how small molecular species, both synthetic as SMDs and biologic in nature as RNA, can contribute to the precision medicine approach along with protein and antibody-based biologics and cell therapies.

Untangling the Complexity of Childhood Atopic Dermatitis: Phenotypes and Endotypes

Dumycz K, Zygadło J, Marzec A, Strus P, Kulus M, Sokolowska M, Venter C, Leung DYM, Feleszko W. Clin Rev Allergy Immunol. 2026 Mar 2;69(1):15. doi: 10.1007/s12016-025-09128-0.

Atopic dermatitis (AD) is a highly prevalent, chronic inflammatory skin disease that predominantly begins in early childhood. Increasing evidence supports that pediatric AD is not a single entity, but rather a spectrum of overlapping phenotypes and endotypes, defined by clinical presentation, immune profile, genetics, ethnicity, and comorbid atopic conditions. This review provides a comprehensive synthesis of current knowledge on childhood AD heterogeneity, emphasizing severity, age of onset, allergen sensitization, allergic comorbidities, ethnicity, and disease trajectory. Phenotypic classification - based on clinical severity, lesion distribution, and disease course - offers valuable prognostic insight, particularly in relation to the risk of asthma, allergic rhinitis, food allergy and eosinophilic esophagitis. Endotyping approaches, including transcriptomic, proteomic, lipidomics, metabolomics and cellular analyses, have revealed distinct immune signatures across age and ethnic groups, including Th2-predominant, Th17/Th22-skewed, or mixed inflammatory pathways. Ethnic-specific differences, such as Th17 activation in Asian populations or Th2/Th22 predominance in individuals of African ancestry, further underscore the need for tailored therapeutic strategies. The emergence of multiomics and clustering-based approaches has begun to identify molecular subgroups, although challenges remain in standardizing methods and validating findings across diverse cohorts. Importantly, the identification of biomarkers to predict disease trajectory and treatment response is a critical unmet need. Future research should prioritize longitudinal, multiethnic, and integrative studies to refine endotyping, enable early intervention, and guide precision medicine in pediatric AD. Understanding the dynamic interplay between genetics, immune dysregulation, and environment will be key to unlocking improved outcomes for children affected by this complex disease.

Davos, May 2026

B Cell Immunology

Dr. Willem van de Veen, PhD

B Cell Immunology Group

Our group investigates how antigen-specific B cell responses are generated, maintained, and reshaped in human disease. Our research focuses on class-switch recombination, antibody subclass diversification, regulatory B cell function, and the differentiation of memory and atypical B cell subsets across allergic and immune-mediated conditions.

We combine high-dimensional immune phenotyping, BCR isotype profiling, plasma proteomics, organoid-based epithelial models, and longitudinal patient cohorts to dissect how B cell-driven immune programs contribute to chronic inflammation, immune tolerance, and therapeutic responsiveness. A central goal of our work is to translate mechanistic insights into biomarker strategies that enable minimally invasive disease monitoring and improved patient stratification.

Over the past year, our projects converged around a common conceptual framework: how B cell compartments adapt and reorganize in response to chronic inflammation or therapeutic intervention.

This included three interrelated research directions:

1. B cells and humoral immunity in eosinophilic esophagitis
We examined systemic and tissue-associated antibody responses, subclass diversification, epithelial barrier stress, and high-dimensional immune signatures distinguishing active and inactive disease. These studies aim to clarify how humoral immunity contributes to disease persistence and remodeling in a chronic type 2 inflammatory setting.

2. Immune monitoring and systems-level profiling
Using integrated mass cytometry, proteomics, and optimized flow cytometry workflows, we defined circulating immune signatures associated with disease activity and therapeutic modulation. In parallel, we contributed to international efforts to standardize outcome definitions and measurement strategies in allergy and clinical immunology, strengthening the translational comparability and reproducibility of immune-based research.

3. B cell remodeling in inflammatory and therapeutic contexts
We investigated how acute allergic reactions, chronic inflammation, and therapeutic immune modulation reshape B cell subsets, antibody responses, and BAFF signaling pathways, including studies in drug hypersensitivity and cancer immunotherapy. These studies highlight how B cell state transitions, ranging from regulatory shifts to expansion of atypical memory populations, are linked to immune tolerance, disease persistence, or therapeutic outcome.

1. B cells and humoral immunity in eosinophilic esophagitis
Circulating Food Allergen-Specific Antibodies, Beyond IgG4, Are Elevated in Eosinophilic Esophagitis
Bel Imam M, Iwasaki S, Lems S, Cevhertas L, Westermann P, Bach Larsen L, Aagaard Poulsen N, Akdis M, Schreiner P, Kreienbühl A, Straumann A, Schoepfer AM, Biedermann L, van de Veen W, Swiss EoE Cohort Study Group.

Clin Exp Allergy. 2025 Oct;55(10):916–927. doi: 10.1111/cea.70055.

Eosinophilic esophagitis has been linked to elevated IgG4, yet it remained unclear whether IgG4 reflects a broader dysregulation of food-specific humoral immunity. We aimed to comprehensively quantify food allergen-specific antibody subclasses to define the systemic humoral signature of EoE and its relationship to disease activity.

We systematically measured food allergen-specific IgG and IgA subclasses across major dietary antigens in patients with active and inactive EoE and in non-EoE controls. Beyond confirming increased IgG4, we observed broader elevations in food-specific IgG subclasses and IgA responses, indicating a complex systemic humoral imprint of EoE. Subclass patterns differed between disease states, suggesting that antibody profiles may reflect underlying inflammatory activity
.
Despite elevated circulating antibodies, allergen-specific B cells were not detectable in peripheral blood, supporting the concept that relevant B cell activation and antibody production may be compartmentalized at the level of the esophageal mucosa. These findings refine the understanding of humoral immunity in EoE and provide a foundation for further tissue-focused investigations.

The Role of B Cells and Antibodies in Eosinophilic Esophagitis
Bel Imam M, Du H, Ardicli Ö, Meinema J, van de Veen W.
Inflamm Intest Dis. 2026. doi: 10.1159/000551065.

EoE is increasingly recognized as a disease not only of eosinophils and type 2 inflammation, but also of tissue remodeling and local immune compartmentalization. This review aimed to integrate emerging evidence on B cells and antibodies in EoE and to define key mechanistic questions for the field.
We synthesize current evidence linking B cells, plasma cells, and antibody responses to EoE pathogenesis. The article integrates data on epithelial barrier dysfunction, type 2 immune pathways, lo-

cal antibody accumulation, and their potential contribution to fibroinflammatory remodeling of the esophagus. The review also discusses how IgG4 biology fits within a broader context of humoral responses and immune regulation.

Key knowledge gaps include the origin and regulation of IgG4-producing plasma cells, the pathways driving local class switching, and how systemic serological findings relate to mucosal immune activity and clinical phenotypes. The review outlines research directions to address these questions and to better connect mechanistic insights with biomarker and intervention strategies.

Systems-Level Immunoprofiling Reveals Distinct Signatures of Active and Inactive Eosinophilic Esophagitis

Bel Imam M, Cevhertas L, Starrenburg ME, Heider A, Bürgi L, Babayev H, Schreiner P, Kreienbühl A, Straumann A, Schoepfer AM, Biedermann L, Swiss EoE Cohort Study Group, van de Veen W.
Under review.

Reliable biomarkers of EoE activity are needed to reduce reliance on repeated endoscopy and to improve disease stratification. We aimed to define circulating cellular and protein signatures that distinguish active from inactive EoE using integrated systems-level immune profiling.

We combined high-dimensional mass cytometry with targeted plasma proteomics to capture coordinated changes across immune cell populations and soluble mediators. We identified significant differences in immune composition between EoE patients and controls, including shifts within $CD4^+$, $CD8^+$, and $CD19^+$ compartments. Within EoE, active and inactive disease states exhibited distinct immune signatures, suggesting measurable systemic correlates of disease activity.

Proteomic analysis revealed a broad set of differentially expressed plasma proteins, including a subset associated with activity. Together, these data provide a framework for developing minimally invasive monitoring strategies and motivate validation in longitudinal cohorts and treatment-stratified studies.

Pathways of Epithelial Barrier Breakdown and Remodeling in Esophageal Organoids by Commonly Used Surfactants

Ardicli Ö, Yazici D, Pat Y, Babayev H, Bel Imam M, Du H, Melhem H, Beha C, Bürgi L, Heider A, Lopez JF, Ardicli S, Zeyneloğlu C, Kreienbühl A, Straumann A, Stocker N, Westermann P, Messner C, Akdis M, Niess JH, Biedermann L, Akdis CA, van de Veen W.
Under review.

Epithelial barrier dysfunction is a central component of EoE, but the contribution of environmental barrier stressors remains incompletely understood. We aimed to test whether commonly used surfactants can induce epithelial injury and remodeling programs in esophageal epithelial models relevant to EoE.

Using patient-derived esophageal organoids and differentiated epithelial cultures, we exposed epithelial tissue models to surfactants under controlled conditions. We observed dose-dependent cytotoxicity and disruption of epithelial integrity, accompanied by activation of inflammatory and remodeling pathways. These findings support the hypothesis that barrier stress can amplify epithelial vulnerability and trigger tissue programs consistent with inflammatory remodeling.

This experimental platform complements our cohort studies by enabling mechanistic interrogation of epithelial barrier disruption under defined exposures and provides a basis for future work linking barrier stress to immune activation pathways.

UpToDate on Eosinophils

Di Gioacchino M, Bagnasco D, Braido F, et al., van de Veen W, Canonica GW.
Exploration of Asthma & Allergy. In press.

Eosinophils are key effector and remodeling-associated cells in a range of allergic and immune-mediated diseases, including eosinophilic gastrointestinal disorders such as eosinophilic esophagitis. This international collaborative review provides an updated synthesis of eosinophil biology and disease-specific functions, with the aim of integrating mechanistic concepts with clinically relevant interpretation.

Within this manuscript, our contribution focused on “Gastrointestinal Diseases”, summarizing current knowledge on eosinophil biology in gastrointestinal inflammatory contexts, including pathways relevant to epithelial barrier dysfunction, tissue inflammation, and remodeling. This section provides context for interpreting eosinophil-associated mechanisms in EoE and supports integration of eosinophil biology with our ongoing work on systemic and tissue-linked immune signatures in eosinophilic gastrointestinal disease.

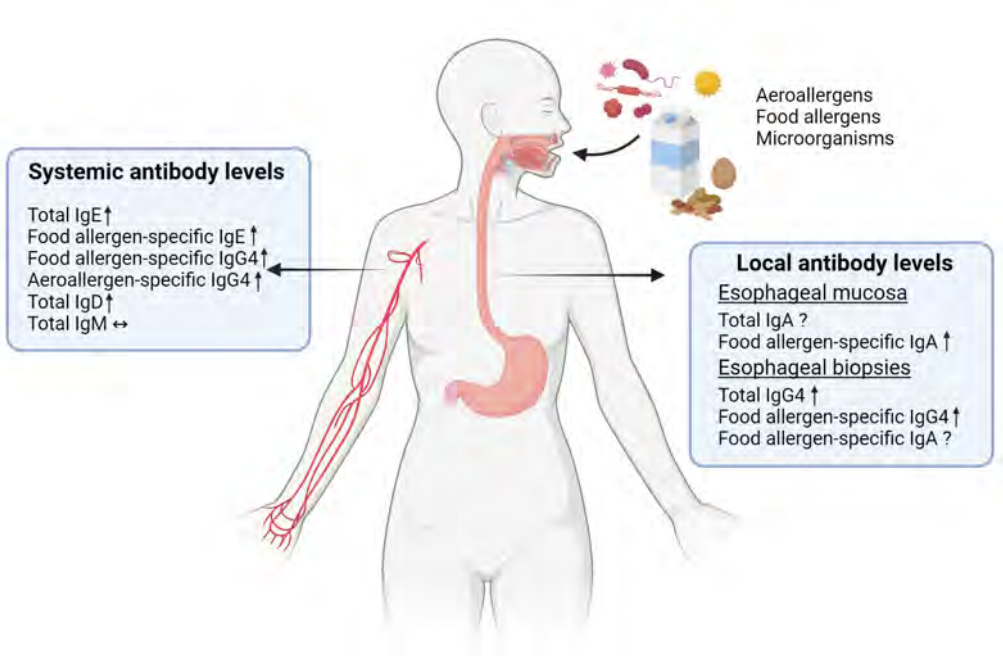


Figure 1 summarizes systemic and tissue-compartmentalized humoral responses in EoE as outlined in Bel Imam et al., 2026 (Inflamm Intest Dis) and provides a conceptual framework for the antibody subclass findings reported in Bel Imam et al., 2025 (Clin Exp Allergy) and our ongoing systems-level profiling studies.

2. Immune monitoring and systems-level profiling

Strategies for Flow Cytometric Profiling of BCR IgH Isotypes: A Comparative Assessment of FcR Blocking Agents
Ardicli O, Lopez JF, Starrenburg ME, Buergi L, Carli TK, Akdis CA, Akdis M, van de Veen W., Allergy. 2025. doi: 10.1111/all.16624.

Precise identification of BCR heavy chain isotypes and subclasses is essential for studying human B cell memory, class switching, and antigen-experienced compartments, but technical variation can bias these measurements. We aimed to systematically assess how Fc receptor blocking strategies affect BCR IgH staining fidelity and to define robust conditions for reproducible profiling.

We compared commonly used Fc receptor blocking approaches under standardized flow cytometry conditions and evaluated their impact on IgH isotype and subclass detection. We show that certain FcR blocking strategies interfere with IgG subclass readouts and can distort interpretation of B cell isotype distributions. The study provides practical recommendations that improve reproducibility and data fidelity.

This methodological work strengthens the foundation of our cohort-based immunophenotyping studies and supports broader standardization efforts in human B cell research.

'What' and 'How' to Measure in Allergy and Clinical Immunology: A Systematic Review of Core Outcome Sets and Outcome Harmonisation Processes
Demidova A, Kiknavelidze N, Purtskhvanidze K, Alieva E, et al., van de Veen W, Genuneit J, Boyle RJ, Apfelbacher C, Munblit D.
Clin Exp Allergy. In press.

Outcome heterogeneity and inconsistent use of measurement instruments limit the comparability and translational value of clinical research in allergy and clinical immunology. We aimed to map existing core outcome sets and harmonisation initiatives and to identify major gaps and methodological limitations across disease areas.

This systematic review demonstrates substantial variation in outcome definitions, measurement instruments, and stakeholder involvement. While some conditions show relatively mature harmonisation efforts, major gaps remain across several allergic and immune-mediated diseases. The work highlights that harmonisation quality is uneven even when initiatives exist.

By providing a structured overview of available core outcomes and harmonisation processes, this study supports improved study design, stronger comparability between trials, and more reliable translation of immunology research into clinical practice.

3. B cell remodeling in inflammatory and therapeutic contexts

Dynamic changes in B cell and immunoglobulin responses after amoxicillin-induced immediate hypersensitivity reactions
Fernandez-Santamaria R, Salas M, Bogas G, Mayorga C, Torres MJ, Akdis CA, Akdis M, van de Veen W.
Under review.

The immune response after acute drug-induced hypersensitivity is dynamic and may evolve over months to years, but these trajectories are not well defined in patients with confirmed immediate reactions. We aimed to longitudinally characterize B cell subset remodeling and drug-specific antibody patterns after amoxicillin-induced immediate hypersensitivity.

We followed patients over two years and assessed trajectories of drug-specific immunoglobulin responses together with changes in B cell and regulatory B cell-related phenotypes. The data indicate time-dependent shifts in antibody subclass patterns and remodeling within memory and regulatory compartments beyond the acute reaction phase. These findings provide insight into post-reaction immune dynamics and may inform optimal windows for immune assessment in diagnostic workflows.

This study contributes to a broader understanding of how acute inflammatory perturbations reshape B cell compartments and how effector and regulatory programs evolve after clinically relevant immune events.

Dupilumab-associated remodeling of B cell memory and IgE responses in pediatric atopic dermatitis
Starrenburg ME, Buergi L, Nguyen NT, Lopez-Crespo JF, Nouwen A, Bel Imam M, Arends NJT, Caspers PJ, Akdis M, Pasmans SGMA, van de Veen W.
J Allergy Clin Immunol. 2024 Nov;154(5):1333–1338.e4. doi: 10.1016/j.jaci.2024.06.023.

Type 2–driven atopic dermatitis is characterized by elevated IgE levels and skewed B cell memory responses. In our previously published study, we demonstrated that systemic IL-4Rα blockade with dupilumab significantly reduced the frequency of type 2 memory B cells (MBC2s), defined as $IgG^{+}CD23^{hi}IL\text{-}4R\alpha^{+}$ B cells, and led to parallel reductions in total IgE levels in pediatric AD patients. These effects were not observed with cyclosporine or topical corticosteroids.

The findings supported the concept that MBC2 cells function as precursors to IgE-producing cells and that IL-4 signaling is central to their maintenance and differentiation. Dupilumab binding to IL-4Rα on both switched and unswitched B cells provided a mechanistic explanation for the observed reduction in IgE class-switch recombination and circulating IgE.

Building on this work, we are currently performing additional high-dimensional analyses on peripheral blood mononuclear cells from the same trial cohort. These include CyTOF-based immune phenotyping to comprehensively map B cell, T cell, and innate immune remodeling under IL-4Rα blockade, as well as targeted and untargeted plasma proteomics to define systemic inflammatory signatures associated with treatment response. These ongoing studies aim to extend the initial mechanistic observations and to identify multidimensional biomarkers of therapeutic response in type 2–mediated disease.

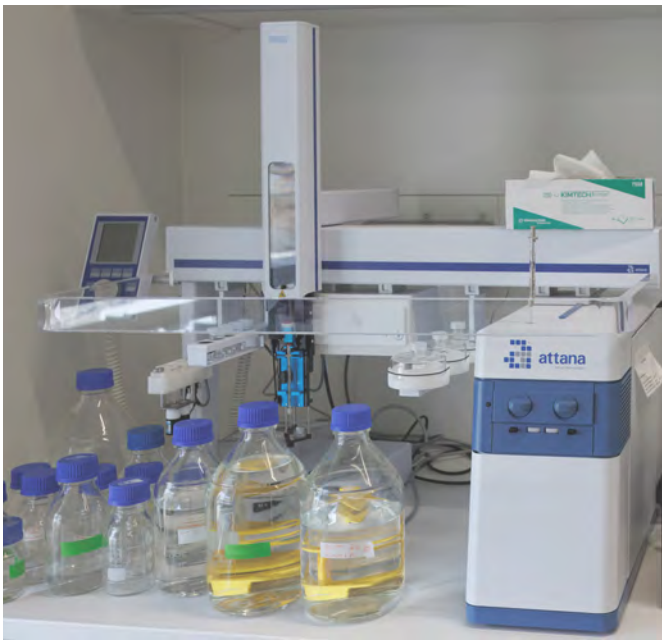
Expansion of $CD21^{-/lo}$ B Cells and BAFF–BAFF-R Axis Dysregulation Associate with Immune Checkpoint Inhibitor Outcome in Metastatic Melanoma
Cevhertas L, Fassler M, Berner F, Bel Imam M, Akdis CA, Akdis M, Flatz L, van de Veen W.
Under review.

Immune checkpoint inhibitors can induce durable clinical responses, yet early biomarkers that predict outcome remain limited, and the contribution of B cell compartment changes is insufficiently understood. We aimed to determine whether circulating B cell subsets and BAFF pathway features associate with response to checkpoint inhibition in metastatic melanoma.

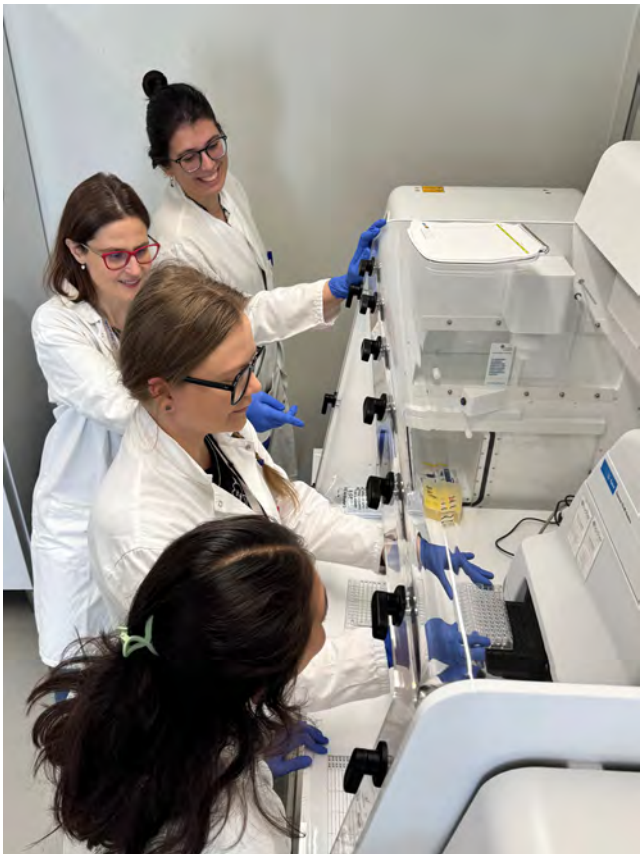
We longitudinally profiled circulating B cell subsets at baseline and during therapy and quantified serum BAFF together with BAFF receptor expression. Non-response was associated with expansion of class-switched CD21-/lo B cells, elevated serum BAFF, and reduced BAFF-R expression. In contrast, responders exhibited higher baseline frequencies of class-switched memory B cells and higher BAFF-R early during therapy, followed by regulated down-modulation over time.

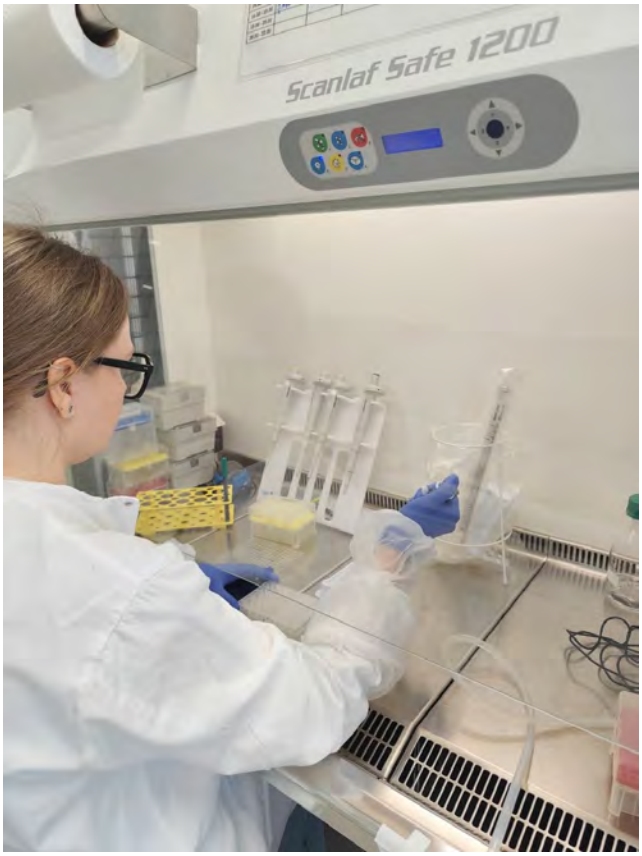
Receiver operating characteristic analysis supports serum BAFF as a potential early discriminator of response. These findings suggest that BAFF signaling dynamics and CD21-/lo B cell expansion are candidate biomarkers for immune monitoring and outcome stratification during checkpoint inhibition.

Davos, May 2026



Laboratory Work





Prof. Dr. C.A. Akdis and Prof. M. Akdis were awarded honorary professorships by the renowned Zhejiang University in China

Award wird nach Cezmi Akdis benannt

Die «European Academy of Allergy and Clinical Immunology» (EAAC I) benennt ihren renommierten Nachwuchsaward neu: Der bisher als «Allergy Award» bekannte Preis trägt künftig den Namen des Davoser Immunologen Professor Cezmi Akdis.

pd | Eine aussergewöhnliche Ehrung für einen aussergewöhnlichen Forscher: Mit der Entscheidung, den prestigeträchtigen «Allergy Award» in «Cezmi Akdis Prize» umzubenennen, würdigt die EAACI das jahrzehntelange Wirken des international anerkannten Immunologen und Direktors des Schweizerischen Instituts für Allergie- und Asthmaforschung (SIAF) in Davos.

Der jährlich verliehene Award würdigt herausragende junge Wissenschaftlerinnen und Wissenschaftler, die mit ihren Arbeiten substanzielle Beiträge zur Allergie- und Immunologieforschung leisten. Ziel ist es, die nächste Generation in der europäischen Forschung zu fördern. Im Rahmen des EAACI-Kongresses in Glasgow wurde am 13. Juni der Award erstmals unter dem neuen Namen «Cezmi Akdis Prize» verliehen.

Vielfältiges Engagement

Professor Cezmi Akdis leitet seit 2007 das SIAF und ist Professor an der Medizinischen Fakultät der Universität Zürich. Er ist Herausgeber des Fachjournals Allergy und wurde für seine wissenschaftlichen Leistungen mit mehreren Ehrendoktorwürden ausgezeichnet. Er ist Mitglied des Senäats der Schweizerischen Akademie der Medizinischen Wissenschaften und hat über 750 wissenschaftliche Publikationen veröffentlicht (H-Index: 169). Mit der von ihm mitgeprägten Epithelbarrieretheorie liefert er zentrale Erklärungen zur Entstehung von Allergien und Autoimmunerkrankungen. Seine Forschung wurde weltweit vielfach zitiert – er wurde unter anderem acht Jahre in Folge in der Liste der «Highly Cited Researchers» von Clarivate geführt. Professor Akdis ist zudem Gründer des internationalen Kongresses «World Immune Regulation Meeting» (WIRM), der seit 2005 jährlich in Davos stattfindet.

Mit der Namensgebung reiht sich der Preis in eine Tradition ein, in der die EAACI herausragende Persönlichkeiten der Wissenschaftsgeschichte ehrt – darunter Paul Ehrlich, Clemens von Pirquet, Daniel Bovet und Charles Blackley. Die Entscheidung für den Namen «Cezmi Akdis» hebt insbesondere seine Beiträge zur Erforschung der Immuntoleranz gegenüber Allergenen, der Mechanismen von Neurodermitis, die erstmalige Beschreibung mehrerer menschlicher Zelltypen sowie seine Epithelbarrieretheorie hervor. Letztere liefert eine Erklärung für den weltweiten Anstieg chronisch entzündlicher, allergischer, autoimmuner und neuropsychiatrischer Erkrankungen.

Dass eine internationale Auszeichnung künftig den Namen eines Davoser Wissenschaftlers trägt, ist nicht nur Ausdruck persönlicher Anerkennung, sondern auch ein starkes Signal für die wissenschaftliche Bedeutung des SIAF auf globaler Ebene.

14 Wissensstadt

Dienstag, 24. Juni 2025
Davoser Zeitung

Proteine – Schlüssel zur Hautgesundheit

Wie neue Technologien Hautkrankheiten aufklären

Das Zentrum für Präzisionsproteomik (ZPP) am Schweizerischen Institut für Allergie- und Asthmaforschung (SIAF) in Davos setzt auf modernste Massenspektrometrie, mit der man die gesamte Zusammensetzung von Proben messen kann. Neueste Technologie ermöglicht die gleichzeitige Messung tausender Proteine, unabhängig davon, ob sich diese im Blut oder in den Hautzellen befinden. Die Daten liefern präzise Einblicke in Entzündungen, Struktur der Haut oder das Ansprechen auf Therapien. Ein besonderes Highlight ist die Nutzung sogenannter schmerzfreier Klebestreifen, mit denen sich winzige Proben direkt von der Hautoberfläche entnehmen lassen. Diese Methode ist besonders geeignet für Kinder und erlaubt eine schonende, grossflächige Datenerhebung – ideal für Studien oder künftige Diagnostik. Davos kann also diese neuen Wege in der Hautforschung dank des Kantons Graubünden gehen, der den Aufbau sowie die Weiterentwicklung dieser hochmodernen Infrastruktur unterstützt hat. Zudem arbeitet das ZPP mit nationalen und internationalen Kliniken und Biobanken zusammen und hat Zugang zu tausenden von Proben. Die Forschung soll zeigen, wie Gene, Hautbarriere und Therapien zusammenwirken, Veränderungen im Proteinhaushalt der Haut auslösen und so Krankheiten mitbestimmen.

Doktorand Philipp Gessner an der Arbeit am Massenspektrometer. Foto: Lopamudra Chatterjee – 21.2.2025

Die Bausteine der Haut verstehen

Proteine übernehmen viele lebenswichtige Aufgaben im Körper. Sie transportieren beispielsweise lebenswichtige Stoffe, welche im Blutkreislauf und innerhalb von Zellen benötigt werden, damit diese richtig funktionieren. Auch der Stoffwechsel, die Hormonproduktion, das Immunsystem oder der Aufbau und die Reparatur von Gewebe werden unter anderem von Proteinen reguliert. Zudem übernehmen sie auch eine Verteidigungsfunktion im Körper, hauptsächlich durch die Produktion von Antikörpern für die Abwehr von Krankheitserregern.

Auch in der Haut sind Proteine unverzichtbar: Sie beeinflussen, wie unsere Haut aussieht, sich anfühlt und auf äussere Reize reagiert. Kein Wunder also, dass sie eine zentrale Rolle bei Hautkrankheiten spielen wie zum Beispiel bei Neurodermitis. Neurodermitis ist eine der häufigsten chronischen Hauterkrankungen, vor allem bei Kindern. Sie verursacht Juckreiz, trockene Haut und Entzündungen und kann die Lebensqualität stark beeinträchtigen. Trotz neuer Medikamente bleiben viele zugrunde liegende Prozesse ungeklärt und verhindern dadurch die gezielte und verbesserte Versorgung von Patientinnen und Patienten sowie eine nachhaltige Steigerung ihres Wohlbefindens.

Forschung, die den PatientInnen zugutekommt

Gemeinsam mit der Dermatologie der Hochgebirgsklinik Davos (HGK) unter der Leitung von PD Dr. med. Matthias Möhrenschlager wird aktuell eine Studie vorbereitet, wie moderne Medikamente für Neurodermitis auf die Haut wirken. Dabei geht es nicht nur um sichtbare Veränderungen in der Haut wie weniger Juckreiz oder Abheilung von Ekzemen, sondern auch um molekulare Veränderungen in der Haut, die durch Veränderungen in den Proteinprofilen abgebildet werden. Die Erkenntnisse sollen helfen, Therapien gezielter und wirksamer einzusetzen. Dank starker Partner wie CK-CARE und der HGK hat sich der Medizincampus Davos zu einem führenden Zentrum für Hautforschung entwickelt. Hier trifft Technologie auf klinische Praxis.

Autoren: Christoph Messner und Philipp Gessner

Das Zentrum für Präzisionsproteomik (ZPP)

Das ZPP wurde 2020 mit dem Entscheid der Regierung des Kantons Graubünden als sogenanntes «Leading House» gegründet. Voll in die Infrastruktur des SIAF eingebettet, fördert das Zentrum wissenschaftliche Exzellenz und enge Zusammenarbeit mit Partnerinstitutionen. Seit 2022 wird das Zentrum von Prof. Dr. Christoph Messner geleitet, der gleichzeitig eine Assistenzprofessur an der Universität Zürich innehat. Obwohl noch jung, ist das Zentrum bereits national wie international anerkannt und arbeitet eng mit Kliniken und Industriepartnern im In- und Ausland zusammen.

PUBLICATIONS

Agache I, Adcock IM, Akdis CA, Akdis M, Bentabol-Ramos G, van den Berge M, Boccabella C, Canonica WG, Caruso C, Couto M, Davila I, Drummond D, Fonseca J, Gherasim A, Del Giacco S, Jackson DJ, Jutel M, Licari A, Loukides S, Moreira A, Mukherjee M, Ojanguren I, Palomares O, Papi A, Perez de Llano L, Price OJ, Rukhazde M, Shamji MH, Shaw D, Sanchez-Garcia S, Testera-Montes A, Torres MJ, Eguiluz-Gracia I. The Bronchodilator and Anti-Inflammatory Effect of Long-Acting Muscarinic Antagonists in Asthma: An EAACI Position Paper. Allergy. 2025 Feb;80(2):380-394. doi: 10.1111/all.16436. Epub 2024 Dec 16. PMID: 39676750 Review. IPF: 12

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SEMINAR AND CONGRESS TALKS

Akdis C. Evidence based forces for life-style changes. EAACI Business Meeting, National Societies. 15 January 2025

Akdis C. Global Health Crisis and Epithelial Barrier Theory. Pediatric Allergy and Asthma Academy. 28 January 2025

Akdis C. Effect of environmental Toxic Substances in Allergy Development. Controversies and Novelties in Allergy. CYNA Congress. Madrid. 31 January 2025

Akdis C. The impact of external exposome on epithelial barriers. American Academy of Allergy Asthma Immunology, AAAAI Congress. San Diego, USA. 28 February 2025

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Akdis C. Akdis C. Epithelial Barrier Theory: Molecular Mechanisms of Tissue Injury. World Immune Regulation Meeting XIX, Davos. 14 March 2025

Akdis C. How to Write and Review High Journal Articles: A Good Reviewer is a Good Author. EAACI Junior Members Online Lecture. 25 March 2025

Akdis C. Epithelial Barrier Theory: Molecular Mechanisms of Tissue Injury. Vumedi online conference. 1 April 2025

Akdis C. Allergic Diseases, Mechanisms, General Overview. Zurich University Course. SIAF, Davos. 1 April 2025

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Akdis C. Epithelial Barrier Theory: Recent Developments and Mechanisms. Zieziyang, Hangzhou University, China. 9 May 2025

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Akdis C. Meet The Experts: How to Make High Impact Publications& How to Review for High Impact Journals. APAAACI Congress, Jakarta, Endonesia. 9-12 October 2025

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Akdis C. Epitleal barrier damage as the mechanism of development of allergic diseases. National Society of Allergy Clinical Immunology Congress, Antalya, Türkiye. 26-30 November 2025

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Messner C. B., High-Throughput Proteomics to Study Human Diseases, CEEPC 2025, Budapest, Hungary, 15.-17. October 2025

Messner C: ProteOmics for Lymphoma and Prognostication, The Loop Annual Meeting, Zurich, 1. Dezember 2025

Messner C: HARMONY: Harnessing Microbial Metabolites for Allergy Prevention and Therapy at Epithelial Barriers,Translationale Medizin an Grenzflächen (TMG), Symposium, Davos, Swizerland, 19 November 2025

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Sokolowska M. Immunometabolic Control of Viral and Allergic Inflammation in Asthma and Allergic Diseases. Discovery 2025. American Academy of Allergy, Asthma & Immunology (AAAI)/ World Allergy Organization (WAO) Joint Congress. San Diego, CA, USA, 28 Feb-03 March 2025

Sokolowska M. Metabolism of Human Th2 Cells in Health and Disease. World Immune Regulation Meeting -XIX, Davos, Switzerland, 12-15 March 2025

Sokolowska M. Breathing with confidence: Journey to Davos and the Science of Healthy Lungs. Academia Raetica Researchers Exchange, Davos, Swizerland, 1 April 2025 (*Public seminar)

Sokolowska M. Restoring the Antiviral Defense in Allergy and Asthma. Romanian Society of Allergology and Clinical Immunology (RSACI) National Conference, Bucharest, Romania 15-17 May 2025

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Sokolowska M. Warum Frauen und Männer anders krank werden? Einblick in die Gendermedizin. Wiiber Hengert 2025, Davos, Switzerland, 20 August 2025 (*Public seminar)

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Sokolowska M. Immunometabolism and Allergy. Asia Pacific Association of Allergy, Asthma and Clinical Immunology (APAAACI) Allergy Week 2025, online, 1-7 September 2025

Sokolowska M. Precision Medicine: From Concept to Clinical Practise. European Academy of Allergy and Clinical Immunology (EAACI) - Clemens von Pirquet Foundation meeting, Kartause Ittingen, Switzerland, 12-13th September 2025

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Sokolowska M. Identifying Key Biomarkers in Allergic Rhinitis and Asthma, FASIT 2025- Allergopharma Expert Meeting, Krakow, Poland, 7-8 November 2025

Sokolowska M. HARMONY: Microbiome-Derived Metabolites in Allergy Prevention, Translationale Medicin an Grenzflächen (TMG) Symposium, Davos, Swizerland, 19 November 2025

Sokolowska M. Metabolic control of epithelial inflammation. Epi-Central Astra Zeneca Expert Exchange, Warsaw, Poland, 21-22 November 2025

Sokolowska M. The role of TSLP in the upper and lower airways. Epicentral United Airways Astra Zeneca Expert Exchange, Zurich, Switzerland, 29 November 2025

Van de Veen W. Role of B Cells in Allergy and Allergen Tolerance. Immunology Lunch Meeting
University Clinic for Rheumatology and Immunology, Bern, Switzerland, 22 May 2025

Van de Veen W. Antigen-Specific Immune Responses in EoE. EAACI Congress 2025, Glasgow, Scotland, 13 - 16 June 2025.

Van de Veen W. Circulating and Cellular Biomarkers of Type 2 Inflammation. OLINK Seminar, University of Zurich, 22-10-2025.

Yazici D. Common food emulsifiers impair epithelial barrier integrity and trigger pro-inflammatory responses. World Immune Regulation Meeting XIX, Davos, Switzerland, 12 – 15 March 2025

Yazici D. Commonly used food emulsifiers disrupt the epithelial barrier and cause a pro-inflammatory response and cell death, 3. Augsburger Neurodermitis-Symposium, Augsburg, Germany, 25. –26. February 2025

Yazici D. Food Emulsifiers Induce Pro-Inflammatory Response and Disrupt the Gut Epithelial Barrier in vitro and in vivo, FOOD ALLERGY FORUM - 4th international conference, Amsterdam, the Netherlands, 22-24 September 2025

CHAIRS AT CONGRESSES

Akdis C. World Immune Regulation Meeting, Session 1. Davos. 12 March 2025

Akdis C. Plenary Session. EAACI Congress, Glasgow, England. 13-16 June 2025

Akdis C. EAACI Journals Symposium. EAACI Congress, Glasgow, England. 13-16 June 2025

Akdis C. Allergen Immunotherapy. National Society of Allergy Clinical Immunology 28 Nove 2025

Akdis C. Meet The Experts Session. APAAACI Congress, Jakarta, Endonesia. 9-12 October 2025

Chairs 2025

Akdis M. OAS: Characterizing Immunity in Inborn Errors of Immunity. AAAAI / WAO Joint Congress, San Diego, USA. February 28-3 March 2025.

Akdis M. SY35 - Updates in the clinical management of allergen immunotherapy. EAACI Congress, Glasgow, England. 13-16 June 2025

Akdis M. SY20 - Present and future of allergen immunotherapy. EAACI Congress, Glasgow, England. 13-16 June 2025

Akdis M. PL5 - Microbiota and Immune Regulation: Insights from Gut to Lungs Across the Lifespan. EAACI Congress, Glasgow, England. 13-16 June 2025

Akdis M. SY5 - Climate change effects on allergic patients: Emerging environmental threats. EAACI Congress, Glasgow, England. 13-16 June 2025

Akdis M. SYM. B Cell-mediated immunity - In memoriam Fritz Melchers. International congress of Immunology IUIS. Vienna, Austria. 17-22 August 2025

Baerenfaller K. Plenary Session 10: Advanced Immune Profiling and Personalized Medicine, World Immune Regulation Meeting (WIRM) 2025, Davos, Switzerland, 12-15 March 2025

Baerenfaller K. [BC]2 Highlights Session, BC2 Basel Computational Biology Conference, Basel, Switzerland, 8-10 September 2025

Baerenfaller K. Keynote Session, BC2 Basel Computational Biology Conference, Basel, Switzerland, 8-10 September 2025

Lopez JF. L-TPS01 - Allergy diagnosis 14. EAACI Annual Congress Glasgow, Scotland, 13-16 June 2025

Lopez JF. TPS28 - Allergy diagnosis 09. EAACI Annual Congress Glasgow, Scotland, 13-16 June 2025

Lopez JF. L-TPS21 - Basic immunology 04. EAACI Annual Congress Glasgow, Scotland, 13-16 June 2025

Ogulur I. Epithelial Barriers, Microbiome and Immune System Interactions. World Immune Regulation Meeting XIX (WIRM-XIX), Davos, Switzerland, 12-15 March 2025.

Ogulur I. Time to redefine healthy eating? EAACI Congress 2025, Glasgow, Scotland, 13-16 June 2025.

Ogulur I. Biologicals, biomarkers and microbiome. EAACI Congress 2025, Glasgow, Scotland, 13-16 June 2025.

Ogulur I. Crosstalk between lung structural cells and immune cells in airway inflammation and remodeling. EAACI Congress 2025, Glasgow, Scotland, 13-16 June 2025

Radzikowska U. Keynote 1 session. Swiss Young Immunologists Society (SYIS) Annual Symposium 2025, Lausanne, Switzerland, 27.08.2025.

Radzikowska U. SYIS Short Communication Session. SSAI Annual Congress 2025, Lausanne, Switzerland, 28-29.08.2025.

Radzikowska U. Poster Session 8: Metabolism. XIX World Immune Regulation Meeting (WIRM), 12-15 March 2025, Davos, Switzerland.

Radzikowska U. Genomics and Proteomics + Ocular Allergy. European Academy of Allergy and Clinical Immunology Annual Congress 2025, 13-16.06.2025, Glasgow, Scotland.

Radzikowska U. Biologicals, biomarkers and microbiome. European Academy of Allergy and Clinical Immunology Annual Congress 2025, 13-16.06.2025, Glasgow, Scotland.

Radzikowska U. Epithelial cell biology and barrier regeneration. European Academy of Allergy and Clinical Immunology Annual Congress 2025, 13-16.06.2025, Glasgow, Scotland

Sokolowska M. 19th introductory course; PhD Program in Microbiology and Immunology (MIM) oral presentation session. Zurich, Switzerland, 20-22 Jan 2025

Sokolowska M. Keynote 1: Muzlifah Haniffa. Human early life development captured in single cell atlases. EAACI Immunology Winterschool, Schladming, Austria, 30 Jan-2 Feb 2025

Sokolowska M. Poster Session II - Topic 4 Microbiome and metabolism. EAACI Immunology Winterschool, Schladming, Austria, 30 Jan-2 Feb 2025

Sokolowska M. Plenary Session Immunometabolism. World Immune Regulation Meeting -XIX, Davos, Switzerland. 12-15 March2025

Sokolowska M. ONE HEALTH-the impact of environment on health plenary session. Romanian Society of Allergology and Clinical Immunology (RSACI) National Conference, Bucharest, Romania 15-17 May 2025

Sokolowska M. PL2 - Infections and the Development of Allergy: Unraveling the Connections. European Academy of Allergy and Clinical Immunology (EAACI) Congress 2024, Glasgow, Scotland 13-16 June 2024

Sokolowska M. TSY1 - Bi-directional neuro-immune communications controlling allergy. European Academy of Allergy and Clinical Immunology (EAACI) Congress 2024, Glasgow, Scotland 13-16 June 2025

Sokolowska M. Allergy Session. Swiss Society of Allergy and Immunology (SSAI) Annual Congress, Lausanne, Switzerland, 28-29 August 2025

Sokolowska M. Oral Abstract Session 5 – Pathophysiology of Allergic Inflammation 2. 34th Biennial Symposium of the Collegium Internationale Allergologicum (CIA), Dubrovnik, Croatia, 27-31 October 2025

Van de veen W. Trained Immunity and Adaptive Immune Responses. World Immune Regulation Meeting, Davos, Switzerland, 12 - 15 March 2025

Van de Veen W. TPS33 - Allergen immunotherapy 04. EAACI Congress 2025, Glasgow, Scotland, 13 - 16 June 2025.

van de Veen W. OAS13 - Diagnosis and management of Eosinophilic Esophagitis, EAACI Congress 2025, Glasgow, Scotland, 13 - 16 June 2025.

van de Veen W. TPS54 - Allergy diagnosis 13, EAACI Congress 2025, Glasgow, Scotland, 13 - 16 June 2025.

Yazici D. Poster Session 5: Epithelial Barrier II, World Immune Regulation Meeting XIX, Davos, Switzerland, 12 – 15 March 2025

Yazici D. Oral Abstract Sessions, Basic Immunology, EAACI 2025, Glasgow, Scotland, 13 – 16 June 2025

Yazici D. Thematic Poster Session, Basic Immunology 02, EAACI 2025, Glasgow, Scotland, 13 – 16 June 2025

Yazici D. Thematic Poster Session, Allergy Diagnosis 17, EAACI 2025, Glasgow, Scotland, 13 – 16 June 2025

DEGREES

Baerenfaller K.
Certificate of Advanced Studies UZH in Hochschuldidaktik, Universität Zürich

AWARDS and GRANTS

Akdis C. Honorary professorship by the renowned Zhejiang University, China, in May 2025.

Akdis C. Honorary professorship from Peking University, China in October 2025.

Akdis C. The EAACI Allergy Award was renamed the Cezmi Akdis Prize.

Akdis M. Honorary professorship by the renowned Zhejiang University, China, in May 2025.

Chatterjee L. Best Oral Presentation Prize. Swiss Proteomics Meeting 2025 in Unterägeri, Switzerland 15-16 May 2025

Egger-Hoerschinger A.-S., Young Investigator Award in Proteomics of the Austrian Proteomics and Metabolomics Association. APMRS 2025, Vienna, 12-13 September 2025

Jardon Parages I. SYIS award for best talk. WIRM, Davos, Switzerland, 12-15 March 2025

Jardon Parages I. Outstanding oral abstract presentation award. EAACI Congress 2025, Glasgow, United Kingdom, 13-16 June 2025

Jardon Parages I. WIRM20 Student–Expert Mixer & Quiz. UZH Alumni Fonds. December 2025

Messner C: SNF Spark; Developing a Highly Sensitive Workflow to Map Antigen-Specific IgE Glycosylation in an Allergic Patient Cohort (237790)

Messner C: The LOOP Zurich BMIP; Clinical proteomics platform for data-driven lymphoma diagnosis and prognostication

Radzikowska U. Best Poster Presentation, EAACI Immunology Winter School 2025, Schladming, Austria, 30.01-02.02.2025

Radzikowska U. Distinguished reviewer for Central European Journal of Immunology 2025

Radzikowska U. SNSF Spark Grant number 229131 "Understanding LUngs: ImmunoMetabOlism of Succinate in asthma (LUMOS)"

Schneider S. Outstanding Oral Abstract Presentation Award. EAACI Allergy School 2025, Cappadocia, Turkyie, 10-12 April 2025

Sokolowska M. Swiss National Science Foundation (SNSF) Individual Research Project Funding Grant nr. 320030-236264

Sokolowska M. Swiss National Science Foundation (SNSF) Lead Agency/Weave Funding Grant nr. 320030E_224154 (with Tomasz Wypych from Polish Academy of Science)

Sokolowska M. Polish National Agency for Academic Exchange (NAWA) Grant (with Wojciech Feleszko from Medical University of Warsaw)

Sokolowska M and Messner C. Translationale Medizin an Grenzflächen (TMG) Research Grant (with Fintan Moriarty, AO Research Institute, Caroline Roduit, Remo Frei and Claudia Traidl-Hoffman from CK-Care and David Niderseer from HGK)

Sokolowska M. OM Pharma Research Grant

Sokolowska M. Roche Consultancy Research Grant

Sokolowska M. Collegium Internationale Allergologicum (CIA) Membership Award and Travel Grant. 34th Biennial Symposium of the Collegium Internationale Allergologicum (CIA), Dubrovnik, Croatia, 27-31 October 2025

Van de Veen W. Alain L. de Weck Travel Grant for the 34th Biennial Symposium of the Collegium Internationale Allergologicum, Dubrovnik, Croatia, 27-31 October 2025

LECTURES

Lectures/Public Seminars 2025

Teaching Activities
(BME 351, BME 364, BME 366, BCH 301):
In 2025, teaching activities comprised contributions to several courses at the University of Zurich and UZH/ETH.

BME 351 – Biomedical Data Mining (3.5 weeks):
Organisation and teaching by PD Dr. K. Bärenfaller, PD Dr. M. Sokolowska, and Prof. Dr. Christoph Messner. The course focused on computational analysis of transcriptomics and proteomics datasets in R, applying a research-based learning approach with student-driven data analysis and hypothesis generation.

BME 364 – Mechanisms of Allergic Diseases (04–26 November 2025):
Teaching by Dr. Ismail Ogulur, PD Dr. M. Sokolowska, Dr. Willem van de Veen, and Dr. Urszula Radzikowska. The course addressed immunological and molecular mechanisms of allergic diseases, including immune sensitisation and memory, type II inflammation, and B Cell biology, complemented by methodological training in data analysis.

BME 366 – Medical Immunology (13 March–03 April 2025):
Teaching by Dr. Willem van de Veen and PD Dr. M. Sokolowska. The course covered fundamental immunological principles with a focus on allergic sensitisation and immune memory.

BCH 301 – Molecular Cell Biology (Immunology)
This lecture has been given for more than 15 years by Drs. Cezmi Akdis and Mübeccel Akdis.
PD Dr. M. Sokolowska contributed four lectures on immunology (24–25 November 2025). Dr. Willem van de Veen contributed a lecture on immune regulation and tolerance (01 December 2025).

Akdis C.
Introduction, Cells, Tissues, Organs
Cell Cooperation in Antibody Response
Hypersensitivity reactions

Akdis M.
Antibodies/B Cells
T Cell Receptors and MHC Molecules

Baerenfalleer K.
BME351 Block Course "Biomedical Data Mining"; responsible PI and main organiser, duration: 3.5 weeks
Lecture in 'Advanced Block Course: Computational Biology' of the Life Science Zurich Graduate School; Topic: Large data sets: Transcriptomics and Proteomics; duration: 3 hours
Lecture in BIO390 'Indroduction to Bioinformatics'; Topic: Proteomics, duration 2 hours
Lectures in the BME364 Block Course: "Statistical analysis and data presentation" and "Multimodal Data Integration in Allergy Research"; duration: 1 hour each

Additional lectures 2025:
636-0704-00L Computational Biology and Bioinformatics Seminar (ETH Zurich), duration: 2 hours
CDS 303 - Data Science und Informatik in der Biologie (University of Applied Sciences of the Grisons FHGR), duration: teaching blocks of 3 x 2 days
Excursion for the talent class Heureka of the Evangelischen Mittelschule Schiers EMS "Von menschlichen Allergien und Bananen"; duration: 2 x 2 hours

Messner C.
BME 364 Mechanisms of allergic diseases. University of Zurich
BME 351 – Biomedical Data Mining. University of Zurich

Ogulur I.
BME 364 Mechanisms of allergic diseases. University of Zurich

Radzikowska U.
UZH/ETH Course BME364 “Mechanisms of Allergic Diseases” and “Biomedical Data Mining”

Sokolowska M.
Innate Immunity
Antigen Presentation
University of Zurich, MNF, BCH301, Molecular Cell Biology, Immunology (24-25.11.2025, 4 lectures)
University of Zurich, MEF, 04SMB3AD, Verdauung, Stoffwechsel, Endokrynologie (15.12.2025, 17.12.2025, 18.12.2025, 4 lectures)
University of Zurich, MNF, BME351 Block course "Biomedical Data Mining" (3 weeks, 04.04.2025-07.05.2025)
University of Zurich, MNF, BME364 Block course "Mechanisms of Allergic Diseases" (3 weeks, 04.11.2025-26.11.2025)
SIAF, Immunology course for Junior Researchers and Master Students

Van de Veen W.
Immune Regulation/Tolerance
BCH301, Fall Semester 2025 Molecular Cell Biology. Immune Regulation/Tolerance 1-12-2025
BME-364, Fall Semester 2025 Mechanisms of Allergic Disease. 04-26 November 2025
Introduction to the immune system
How to analyze Flow cytometry data
Type II Inflammation - Role of B Cells
BME-366, Spring Semester 2025 Medical Immunology. 13 March - 03 April 2025
Allergic sensitization and immune memory in allergy

PUBLIC SEMINARS

08.01.2025 – Prof. Dr. med. Dr. h. c. Torsten Zuberbier
Charité – Universitätsmedizin Berlin, Institute of Allergology IFA, Fraunhofer ITMP
Immunology and Allergology IA, Berlin, Germany
Title: “Molecular mechanims of different types urticaria"

04.02.2025 – PD Dr. Akos Dobay

Co-head of the Forensic Machine Learning Technology Center (ForMaLTeC) Zurich, Zurich Institute of Forensic Medicine, University of Zurich, Switzerland
Title: “The Transformative Impact of AI on Forensic Science"

20.02.2025 – Dr. Rafael Argüello
Aix Marseille Univ, CNRS, INSERM, CIML, Centre d’Immunologie de Marseille-Luminy,
Marseille, France
Title: “Approaching Human ImmunoMetabolism in the Single-Cell Era"

10.03.2025 – Dr. sc. nat. Jan Wohlfahrt
European and German patent attorney and partner at Gleiss Große Schrell und Partner mbB, Stuttgart, Germany
Title: “From clones to claims, from proteins to patents - Intellectual property in biotech and medicine"

10.03.2025 – Dr. Ganesh Pandian Namasivayam
Head and Principal Investigator, Intelligent ChemBioInformatics (INCBI), Kyoto University, Japan
Title: “Flipping the Switch: Exploring Mitochondrial Immunotherapy for Space Travel Adaptation and Asthma Research"

28.03.2025 – Prof. Dr. Onur Boyman
Director, Department of Immunology, University Center for Laboratory Medicine and Pathology
UZL), University of Zurich, Switzerland
Title: “The Transformative Impact of AI on Forensic Science"

10.06.2026 - Prof. Franca Ronchese
Programme Leader, Malaghan Institute of Medical Research, Wellington, New Zealand
Title: “How allergies start in the skin"

14.07.2025 - Prof. Ali Önder Yildirim
Institute of Lung Health and Immunity, Helmholtz Munich, Germany
Title: “Precision immunology for chronic lung diseases"

30.09.2025 - Prof. Dr. Peter Wick
Head Nanomaterials in Health, Adj Professor D-HEST, ETH Zurich, Empa, Swiss Federal Laboratories for Materials Science and Technology
St. Gallen, Switzerland
Title: “Micro- and Nanoplastics (MNPs): A women's and maternity health perspective"

22.10.2025 – SIAF/Olink Seminar
Carina Beha – SIAF’s presentation
Lutz Priebe – Olink technology
Shafeeq Mohammed – Epigenetic Editing of Chromatin Readers Rescues Doxorubicin-Induced Endothelial Senescence and Vascular Dysfunction
Willem Derk Van de Veen– Circulating and Cellular Biomarkers of Type 2 Inflammation
Martina Konantz – Biomarkers in Mastocytosis
Miriam Rabl – Brain inflammatory processes underlying neuropsychiatric symptoms in older people

SIAF SCIENCE DAY
17.12.2025

Lopamudra Chatterjee “MasterChef: Lymphoma Edition”
Paolo D`Avino, “Skin Game”
Hang Du, “A Part-Time Vampire, Full-Time PhD Student”
Anna-Sophia Egger, “Sherlock Holmes and the Case of the Mysterious Metabolome”
Philipp Gessner, “Skin Proteins - Gotta Catch'em All!”
Tanya Guevara, “Gut Feeling: How Microbes Power Your T Cells”
Ines Jardon Parages, “The Winter MetabOlympics: May the Best Cell Win!”
Angel Maldonado, “Get high”
Xindong Sun, “Don't let glycan go!”
Minglin Yang, “Mario’s Dream Quest: Discovering HR1 and HR2 Functions in B Cells”
Can Zeyneloglu, “Small Additive, Big Attitude: P80’s Journey from Epithelial Chaos to Systemic Inflammation”
Bingjie Zhao, “A story about protecting my intestine city"

Winner of the SIAF Science Day 2025:
Paolo D`Avino

SCIENTIFIC POSTS AND EDITORIAL ACTIVITIES

Akdis CA.
Allergy, Editor in Chief
Current Opinion in Immunology, editorial board member
Expert Opinion on Emerging Drugs, editorial board member
International Reviews of Immunology, editorial board member
Journal of Investigational Allergology and Clinical Immunology, editorial board member
American Academy of Allergy, Asthma & Immunology (AAAAI) - Eczema Atopic Dermatitis Committee Member
American Academy of Allergy, Asthma & Immunology (AAAAI) - Cells and Mediators Committee, Board Member
Christine Kuehne - Center for Allergy Research and Education (CK-CARE) – Directorium member
COST Action BM0806 - Recent advances in histamine receptor H4 research member
National Institute of Health, USA - Scientific Advisory Board, Food Allergy, Allergen-Specific Immunotherapy

Scientific Posts and Editorial Activities 2025

European Academy of Allergy Clinical Immunology (EAACI) – Member of Biologicals Guidelines
European Academy of Allergy Clinical Immunology (EAACI) - Member of Allergen Immunotherapy Guidelines
EAACI Research and Outreach Committee (ROC) Immunology Chair
European Asthma Research and Innovation Partnership (EARIP) - Member
Global Allergy and Asthma European Network GA2LEN - Member
World Immune Regulation Meeting - Chairman
Stanford University, School of Medicine, Department of Immunology, Sean Parker Allergy Center - Scientific Advisory Board Member

Akdis M.
Principal Investigator-The Microbiology and Immunology PhD program, UZH-ETH
EAACI Research and Outreach Committee (ROC) Member
EAACI Food Allergy Guidelines member
European Academy of Allergy Clinical Immunology (EAACI) – Member of Biologicals Guidelines
Member of Scientific Board of Sean Parker Allergy Center, Stanford
Member of Scientific Board of Leo Foundation Skin Immunology Research Center
Workpackage Member of EU project CURE
Allergy, Editorial Board Member
Journal of Allergy and Clinical Immunology, Reviewer Board Member
SNF project reviewer
PAI, Reviewer Board Member
Science Foundation Ireland, Reviewer Board member
World Immune Regulation Meeting, Member of the scientific committee

Baerenfaller K.
Faculty Member with the Right to Confer a PhD at the Science Faculty of the University of Zurich
Principal Investigator – Systems Biology PhD program (UZH/ETH)
Principal Investigator – Molecular Life Science PhD program (UZH/ETH)
Member of the Scientific Committee of the World Immune Regulation Meeting (WIRM)
Member of the Scientific Committee Core Team (SCCT) of the BC2 Basel Computational Biology Conference
Member of the SIB Board of Directors
Group leader of the Swiss Institute of Bioinformatics (SIB)
Member of LS2, President of the LS2 Bioinformatics intersection
Member of the European Reference Genome Atlas (ERGA)
Member of EAACI (European Academy of Allergy & Clinical Immunology)
Board member of Science City Davos
Project leader of ETH Studio Davos
President of Naturforschende Gesellschaft Davos
Member of the school board of Alpine Mittelschule Davos (SAMD)

Messner C.
Assistant Professor for Precision Proteomics – Medical Faculty, University of Zurich
Principal Investigator- Systems Biology PhD program (UZH /ETH)
Principal Investigator- Molecular Life Sciences PhD program (UZH/ETH)
Principal Investigator-The Microbiology and Immunology PhD program (UZH/ETH)
Group leader of the Swiss Institute of Bioinformatics (SIB)
Member of the technical board of the Proteomics Unit of the Functional Genomics Center Zurich (FGCZ) (ETH/UZH)
Member of the Comprehensive Cancer Center Zurich (CCCZ), University hospital Zurich
Member of EAACI (European Academy of Allergy & Clinical Immunology)
World Immune Regulation Meeting, Member of the scientific committee
Member of Life Science Switzerland, Proteomics and Bioinformatics Section
Scientific advisory board for the ETH Zurich Pioneer Fellowship
Expert Reviewer for Grant agencies: Austrian Science Fund (FWF), Pancreatic Cancer UK
Reviewer for Nature Communications, Nature Protocols, Nature Biomedical Engineering, ACS omega, Expert review of proteomics, Allergy, Journal of Proteome Research, Nucleic Acids Research, Trends in Analytical Chemistry

Ogulur Ismail
Chair, European Academy of Allergy and Clinical Immunology (EAACI), Epithelial Cell Biology Working Group
Frontiers in Allergy, Guest Associate Editor
Scientific referee for peer-reviewed journals: Allergy, Gut Microbes, Ecotoxicology and Environmental Safety, Inflammation, Biomolecules, Nutrients, Food and Chemical Toxicology, Vaccine

Rhyner C.
Allergy, member of the editorial board
JACI, Member of the reviewer board
Int Arch All, member of the editorial board
Frontiers in Allergy Therapies, Therapeutic targets and Mechanisms, Associated Editor
EAACI Interest Group „Clinical and veterinary allergology", member of the board
Member of Life Sciences Zurich Graduate School-Zurich
World Immune Regulation Meeting, Member of the scientific committee

Sokolowska M.
Principal Investigator- the Microbiology and Immunology PhD program, UZH-ETH
Delegate- UZH, Faculty of Medicine, Basic Science Committee (FB2)
Chair, European Academy of Allergy and Clinical Immunology (EAACI) Basic and Clinical Immunology Section
Chair, European Academy of Allergy and Clinical Immunology (EAACI) Research and Outreach Committee (ROC), Immunology Working Group

Chair, European Academy of Allergy and Clinical Immunology (EAACI) Task Force on Immune Metabolism
Secretary, European Academy of Allergy and Clinical Immunology (EAACI) Task Force on Eicosanoids
Secretary, European Academy of Allergy and Clinical Immunology (EAACI) Task Force on Public Outreach
Chair, European Academy of Allergy and Clinical Immunology (EAACI) Immunology Winterschool 2024, Zakopane, Poland
Co-Chair, World Immune Regulation Meeting 2024
Chair, International Symposium on Molecular Allergology- European Rhinallegy Meeting, ISMA-RHINA 2024, Helsinki, Finland
Expert Reviewer for Grant agencies: Medical Research Council, UK Research and Innovation (MRC-UKRI); German Research Foundation (DFG), Estonian National Research Foundation, Alexander von Humboldt Foundation, Joint Excellence in Science and Humanities (JESH), Austrian Academy of Sciences, National Science Centre (NCN) Poland, and Vrije Universiteit Brussels (VUB)
Editorial activities:
Heliyon, Cell Press, Associate Editor Immunology
Scientific Reports, Editorial Board member
Allergy, Editorial Board member
Frontiers in Immunology, Editorial Board member
Clinical and Molecular Allergy, Editorial Board member
Frontiers in Pharmacology, Reviewer Board member
Frontiers in Allergy, Reviewer Board member
Scientific referee for peer-reviewed journals: Immunity, New England Journal of Medicine, Nature Chemical Biology, Allergy, Journal of Allergy and Clinical Immunology, European Respiratory Journal, Scientific Reports, Antioxidant and Redox Signaling, Plos One, Signal Transduction and Targeted Therapy.

van de Veen W.
Editorial board – Immunotherapy Advances
Associate editor – Frontiers in Allergy
Editorial board member - Exploration of Asthma & Allergy
Member College of Expert Reviewers - European Science Foundation (ESF).
Management committee member - COST action entitled: “The Core Outcome Measures for Food Allergy”.
Programme committee member - the Graduate School Graubünden.
Reviewer board member - Journal of Allergy and Clinical Immunology (JACI), Frontiers in Allergy.
Editorial board member - Allergy
Scientific referee for peer-reviewed journals: Journal of Allergy and Clinical Immunology (JACI), Allergy, PLOS ONE, Nature Scientific Reports, Journal of Investigational Allergology and Clinical Immunology (JIACI), European journal of immunology (EJI), International Archives of Allergy and Immunology, Immunotherapy Advances, Frontiers in Immunology, Frontiers in Allergy, npj vaccines, Science Immunology.
Co-Chair – World Immune Regulation Meeting
Memberships:
European Academy of Allergy and Clinical Immunology (EAACI)
British Society for Immunology (BSI)

NATIONAL AND INTERNATIONAL COLLABORATIONS

Amsterdam UMC, Department of Pulmonary Medicine, (NL), Dr. E Weersink, Prof S Vijverberg
Allergopharma GmbH & Co. KG., Reinbek (DE), Dr. A. Nandy, Dr. C. Willers, Dr. H. Kahlert, Dr. N. Berek

Allgem. Krankenhaus (AKH) Wien (AT), Institut für Allgemeine und Experimentelle Pathologie, Prof. H. Breiteneder, Dr. P. Ebersteiner, Prof. E.-J. Jarolim, Dr. S. Natter, Prof. O. Scheiner, Prof. R. Valenta, Dr. S. Vrtala

AO Research Institute Davos, (CH), Dr. S. Grad, Prof. M. Alini, Dr. F. Moriarty, Prof. R.G. Richards, Dr. K. Thompson, Prof. M. Stoddart, Prof. B. Gueorguiev, Dr. J. Barcik, Dr. T. Serra, Prof. M. D'Este, Dr. E. Della Bella

Beckman Research Institute, Department of Molecular and Cellular Biology, City of Hope (US), Dr. M. Boldin

Benaroya Research Institute at Virginia Mason; Department of Medicine, University of Washington (US), Dr. W. Kwok, IT Chow

Bilkent University, Ankara (TR), Prof. I. Gürsel

Biognosys AG, Schlieren, Dr. Nigel Beaton, Dr. M. Tognetti

Bispebjerg Hospital, Copenhagen University, Denmark, Dr. Christian Woehlk, Prof. Celeste Porsbjerg

Charité - Universitätsmedizin Berlin, Institut für Biochemie, Prof. M. Ralser

Center for Inflammation Research, University of Edinburgh (UK), Prof. J. Schwartze

Cantonal Office for Nature and Environment of the Grisons, Chur (CH); Cantonal Office for Food

Security and Animal Health of GR, Cantonal Office for Anture and Environment, Cantonal for Military and Civil Protection, Cantonal office for Health

Centre d'Immunologie de Marseille-Luminy, Marseille, France, Dr. Rafael J. Argüello

Centre Suisse d‘Electronique et Microtechnique SA (CSEM)
Landquart (CH), Dr. S. Generelli, Dr. D. Ulrich

Complutense University Madrid (ES), Dr. O. Palomares, Dr. M. Martin-Fonseca, Dr. A. Querencias

Consejo Superior de Investigaciones Cienti cas (CSIC), Madrid (ES), Dr. C. Bernabéu

CURE partners: Prof. N. Papadopoulous, Assistant Prof. P. Xepa- padaki, Dr. S. Taka, Assistant Prof. N. Rovina, Prof. D. Robertson, Dr. T. Gilman, Dr. S. Megremis, Dr. E. Andreakos, Prof. KB. Marcu, Dr. I. Galani, Prof. ML. Kowalski, Prof. X. Thibert-Plante, Dr. N.
Cahnishivili, Dr. M. Goderdzishvili, G. De Carlo

Department of Food Science, Aarhus University (DK), Prof. L. Bach Larsen, Prof. N. Aagaard Poulsen

Department of Infectious Diseases and Hospital Epidemiology, University Spital Zurich, CH, Prof. Silvio Brugger, Dr. Weronika Barcik

Endophyte Service Laboratory in Corvallis (US), Dr. Jenni Duringer

Erasmus MC, Rotterdam (NL), Dr. R. de Swart, Prof. S. Pasmans, Dr. M. Schreurs

National and International Collaborations 2025

ETH Zürich (CH), Computational Systems Biology Group, Prof. J. Stelling; Institute of Pharmaceutical Sciences, Prof. G. Folkers; Department of Biosystems Science and Engineering, DS. Roqueiro; ETH Studio Davos
Forschungszentrum Borstel (DE), Prof. U. Jappe, Prof. H. Fehrenbach, Prof. Dr. O. Holst

ETH Zurich, CH Prof. Dr. Nicola Zamboni, Institute of Molecular Systems Biology,
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Schweizerisches Institut für Allergie- und Asthmaforschung

Bilanz per 31. Dezember 2025

(inklusive Drittmittel)

	31.12.2025	31.12.2024
	CHF	CHF
AKTIVEN		
Flüssige Mittel	1'037'322.33	714'607.43
Forderungen	253'191.99	235'469.64
Kontokorrent SFI Stiftung	78'794.20	28'542.45
Aktive Rechnungsabgrenzungen	623'541.33	513'274.60
	1'992'849.85	1'491'894.12
PASSIVEN		
Verbindlichkeiten	851'069.56	609'638.78
Kontokorrent PMOD/WRC	0	5'939.15
Passive Rechnungsabgrenzungen	1'045'874.41	760'188.88
Rückstellungen	0	20'221.43
Eigenkapital	95'905.88	95'905.88
	1'992'849.85	1'491'894.12

Schweizerisches Institut für Allergie- und Asthmaforschung

Betriebsrechnung 2025

(inklusive Drittmittel)

	Abschluss 2025	Budget 2025	Abschluss 2024
	CHF	CHF	CHF
ERTRAG			
Beitrag Bund Forschungsgesetz Art. 15	1'500'000.00	1'292'400.00	1'292'400.00
Beitrag Kanton Graubünden	1'049'999.70	1'048'000.00	780'000.00
Beitrag Kanton Graubünden Sonderprofessur "Precision-Proteomics-Center"	540'064.70	0	755'483.28
Beitrag Gemeinde Davos	524'560.00	524'560.00	524'560.00
Beitrag Universität Zürich	389'818.00	392'063.00	387'704.70
Beitrag Stiftung SFI	79'500.00	79'500.00	21'618.60
Beitrag Stiftung vormals Bündner Heilstätte Arosa	57'546.00	57'546.00	56'108.50
Beitrag Stiftungen/Drittmittel	29'194.46	0	0
Overheadbeiträge	19'419.90	0	18'564.00
Patenterlöse	180'385.00	0	253'125.78
Übriger Ertrag	7'722.36	3'000.00	41'879.53
Finanzertrag	0	6'650.00	0
Ausserordentlicher Ertrag	18'631.72	35'000.00	33'710.98
Auflösung von Rückstellungen	20'221.43	0	175'478.84
WIRM-Kongress	214'103.37	259'565.00	228'267.34
Drittmittel	1'930'115.64	2'663'706.00	2'980'899.75
	6'561'282.28	6'361'990.00	7'549'801.30
AUFWAND			
Personalaufwand	3'429'978.30	3'598'612.00	3'931'745.63
Verbrauchsmaterial	1'352'815.14	1'279'420.00	1'691'878.17
Raumaufwand	317'917.78	341'000.00	312'278.74
Unterhalt/Reparaturen/Ersatz	488'888.29	346'000.00	435'103.59
Investitionen	177'236.76	75'000.00	309'018.88
Sachversicherungen/Abgaben	20'159.20	20'000.00	18'106.80
Energie- und Entsorgungsaufwand	186'995.27	202'000.00	166'532.75
Verwaltungsaufwand	149'995.39	107'500.00	171'227.99
Werbeaufwand	18'374.01	125'000.00	17'203.52
Reisespesen	97'381.48		148'957.44
WIRM-Kongress	159'888.10	205'500.00	170'843.46
Übriger Betriebsaufwand	15'851.45	11'000.00	28'311.39
Abschreibungen	105'200.00	105'200.00	105'200.00
Finanzaufwand	2'936.58	4'000.00	473.56
Ausserordentlicher Aufwand	37'664.53	1'000.00	42'919.38
	6'561'282.28	6'421'232.00	7'549'801.30
Ergebnis	0.00	-59'242.00	0.00
	6'561'282.28	6'361'990.00	7'549'801.30

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