Prof. Dr. Cezmi A. Akdis

Swiss Institute of Allergy and Asthma Research

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Index

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 arised 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

Lecture to Zurich University Sutendts in SIAF on Epithelial Barriers 2024

Bericht des Direktors

Prof. Dr. Cezmi A. Akdis

Das Schweizerische Institut für Allergie- und Asthmaforschung (SIAF), gegründet 1988 von der Medizinischen Abteilung des Schweizerischen Forschungsinstituts für Hochgebirgsklima und Medizin Davos (SFI), hat das Ziel durch seine Forschung das Verständnis von Allergien und Asthma zu verbessern. Das Ziel ist es, Behandlungen zu entwickeln, die die Zukunft der Betroffenen verbessern. Seit 1996 ist das SIAF der Universität Zürich angeschlossen und seit 2008 Mitglied der Life Science Zurich Graduate School, einem gemeinsamen Bildungsprojekt der Universität Zürich und der ETH Zürich. Diese Zugehörigkeit ermöglicht es dem SIAF umfassende PhD-Ausbildungen anzubieten. Darüber hinaus ist das SIAF ein aktives Mitglied der Academia Raetica und der Graduate School des Kantons Graubünden.

Forschung am SIAF ist so strukturiert, dass sie eine enge Zusammenarbeit mit klinischen Einrichtungen in Davos, der Universität Zürich und weiteren spezialisierten Forschungszentren fördert. Das Institut widmet sich der patientenzentrierten translationalen Forschung mit einem klaren Schwerpunkt auf der Aufklärung immunologischer Mechanismen, die allergischen und asthmatischen Erkrankungen zugrunde liegen. Ziel dieser Arbeiten ist die Entwicklung innovativer präventiver und therapeutischer Strategien für betroffene Personen.

Das SIAF engagiert sich aktiv im Austausch mit führenden internationalen Organisationen wie der European Academy of Allergy and Clinical Immunology (EAACI), der American Academy of Allergy, Asthma & Immunology (AAAAI) und der World Allergy Organization (WAO). Zudem pflegt es enge Kooperationen mit renommierten akademischen Institutionen wie dem Sean Parker Center for Asthma and Allergy Research der Stanford University und der T.H. Chan School of Public Health der Harvard University.

Darüber hinaus baut das Institut kontinuierlich ein Team von Bioinformatikerinnen und Bioinformatikern auf und integriert zunehmend Ansätze der Künstlichen Intelligenz und des Maschinellen Lernens in seine Forschung. Mehrere fortgeschrittene Projekte befinden sich derzeit in der Umsetzung.

Im Jahr 2024 wurden 78 wissenschaftliche, durch Gutachter geprüfte Arbeiten in internationalen Zeitschriften mit einem Impact-Factor veröffentlicht. Das SIAF erreichte im Jahr 2024 einen **Gesamt-Impact-Faktor-Wert von 906.094** und einen **Durchschnitt von 11.617 Punkten** pro Veröffentlichung. Die neuesten Erkenntnisse wurden zudem in **70 Abstracts** auf verschiedenen Fachkonferenzen präsentiert. Unsere Mitarbeiter wurden eingeladen, an **123** verschiedenen Seminaren und Präsentationen auf nationalen und internationalen Kongressen teilzunehmen. Solche Einladungen sind wichtig für die Verbreitung der erzielten Ergebnisse und für die internationale Akzeptanz der Forschungsarbeit des Instituts. SIAF-Mitarbeiter leiteten **37** verschiedene Sitzungen. Darüber hinaus bekleiden SIAF-Mitarbeiter **67** wissenschaftliche Positionen in internationalen Gesellschaften und **34** Positionen in internationalen Zeitschriften. Seit 2018 ist Prof. C.A. Akdis Chefredakteur der Zeitschrift Allergy. Unter seiner Leitung stieg der Impact-Faktor des Fachjournals von **6.02 auf 12.6**, womit sie zur führenden Zeitschrift im Bereich der Allergie- und Klinischen Immunologie wurde.

Aufgrund der international hoch angesehenen wissenschaftlichen Publikationen wurde Prof. Dr. C.A. Akdis 2024 zum neunten Mal von Thomson Reuters Clarivate in die Liste der meistzitierten Forscher aus allen wissenschaftlichen Disziplinen weltweit aufgenommen. Im Jahr 2024 wurde PD Dr. Milena Sokolowska, Leiterin der Immune Metabolism Group am SIAF, erstmals in diese Liste aufgenommen. Das SIAF hat rund 1.783 wissenschaftliche Beiträge veröffentlicht und gehört zu den meistzitierten Instituten weltweit. Die vom SIAF veröffentlichten Artikel wurden mehr als 97‘000 mal zitiert..

Die von Cezmi Akdis formulierte **Epithelbarrieretheorie** besagt, dass Schädigungen der epithelialen Oberflächen des Körpers – wie Haut, Atemwege und Darmschleimhaut – eine zentrale Rolle bei der Entstehung zahlreicher chronischer Erkrankungen spielen, darunter Allergien, Asthma, Autoimmunerkrankungen, Stoffwechselstörungen und sogar bestimmte neuropsychiatrische Erkrankungen. Epithelbarrieren bilden die erste Verteidigungslinie des Körpers gegenüber Umweltfaktoren, wie Krankheitserregern, Schadstoffen, Allergenen und Toxinen. Solange diese Barrieren intakt sind, verhindern sie das Eindringen schädlicher Substanzen in tiefer liegende Gewebe und das Auslösen von Immunreaktionen.

Moderne Lebensweisen – geprägt durch vermehrte Belastung mit verarbeiteten Lebensmitteln, Reinigungsmitteln, Mikroplastik, Luftverschmutzung und Antibiotika – können jedoch die Integrität dieser Barrieren beeinträchtigen. Dies führt zu einer erhöhten Durchlässigkeit („Leaky Barriers“), wodurch Mikroben und Toxine in tiefere Gewebeschichten eindringen können. In der Folge entstehen chronische Entzündungen und eine fehlgesteuerte Immunaktivierung. Neue Forschungsergebnisse zeigen, dass gestörte Epithelbarrieren nicht nur Folgeerscheinungen, sondern möglicherweise zentrale Auslöser der Krankheitsentstehung sind – insbesondere bei genetisch anfälligen Personen.

Die Theorie hebt auch die bedeutende Rolle des Mikrobioms hervor, das in enger Wechselwirkung mit den Epitheloberflächen steht und zur Aufrechterhaltung der Immuntoleranz beiträgt. Das Verständnis und der Schutz der Epithelbarriere gelten daher als vielversprechender Ansatz zur Krankheitsprävention und zur Entwicklung neuer, wirksamerer Therapien für eine Vielzahl chronischer Erkrankungen.

Laut der Epithelbarrieretheorie können **moderne Umweltfaktoren** wie **Reinigungsmittel, Luftverschmutzung** und **Lebensmittelzusatzstoffe** – insbesondere **Emulgatoren und Konservierungsstoffe** – die Integrität epithelialer Oberflächen beeinträchtigen und zur Entstehung chronischer, entzündlicher und immunvermittelter Erkrankungen beitragen.

Reinigungsmittel und Putzmittel, die im häuslichen und öffentlichen Bereich weit verbreitet sind, enthalten Tenside und andere chemische Substanzen, die die Epithelbarriere der Haut und der Atemwege schädigen können. Häufige Exposition schwächt die Zellverbindungen (Tight Junctions) zwischen Epithelzellen, erhöht die Durchlässigkeit und ermöglicht das Eindringen von Allergenen, Krankheitserregern und Toxinen in tiefere Gewebeschichten. Be-

Prof. Dr. Cezmi A. Akdis

sonders gefährdet sind Säuglinge und Sportler, da sie häufiger mit solchen Substanzen in Kontakt kommen – etwa durch Hygienemaßnahmen oder Umwelteinflüsse.

Luftverschmutzung, einschließlich Feinstaub (PM2.5 und PM10), Dieselabgasen, Ozon und Stickstoffdioxid, beeinträchtigt nachweislich die Epithelbarriere der Atemwege. Diese Schadstoffe verursachen oxidativen Stress und Entzündungen, schädigen die Zellmembranen der Epithelzellen und verändern die Struktur von Tight Junctions. Dadurch steigt nicht nur das Risiko für Atemwegsinfektionen und Asthma, sondern auch für systemische Immunreaktionen.

Lebensmittelzusatzstoffe, wie Emulgatoren, künstliche Süßstoffe und Konservierungsstoffe, stehen im Zusammenhang mit einer erhöhten Durchlässigkeit der Darmschleimhaut. Emulgatoren wie Polysorbat 80 und Carboxymethylcellulose stören die Schleimschicht des Darms und das Gleichgewicht des Mikrobioms, was zu chronischer Entzündung, mikrobieller Dysbalance (Dysbiose) und Stoffwechselstörungen führen kann. Dadurch wird das Eindringen von Bakterien und Endotoxinen in den Blutkreislauf begünstigt, was unter anderem zur Entstehung von Adipositas, chronisch-entzündlichen Darmerkrankungen und anderen Leiden beitragen kann.

In ihrer Gesamtheit untergraben diese modernen Umweltbelastungen die natürlichen Schutzbarrieren des Körpers, fördern fehlgesteuerte Immunantworten und könnten eine Schlüsselrolle beim weltweiten Anstieg allergischer, autoimmuner und metabolischer Erkrankungen spielen.

Precision Proteomics Center: Eines der Schlüsselprojekte des Instituts ist die Einrichtung des Precision Proteomics Center am SIAF in Zusammenarbeit mit dem Kanton Graubünden und der Universität Zürich. Das Zentrum wurde erfolgreich eingerichtet und befindet sich nun unter der Leitung von Prof. Christoph Messner in der Phase der Personal- und Infrastrukturerweiterung. Das Zentrum entwickelt und wendet modernste massenspektrometrie-basierte Technologien für die Proteomanalyse klinischer Proben an. Ziel des Zentrums ist es, neue Biomarker und Krankheitsmechanismen zu identifizieren, die zur Entwicklung der nächsten Generation personalisierter Behandlungen beitragen werden.

· Etablierung von Hochdurchsatz-Proteomik-Technologien für Anwendungen in der Biomarker-Entdeckung bei Allergien, Hautkrankheiten und Onkologie.

· Neue Technik 'OxoScan-MS' für grossangelegte Plasma-Glycoproteomik: Wir haben eine Methode entwickelt, die eine quantitative Kartierung von Glykopeptiden in großen Kohorten ermöglicht und eine differenzielle Glycosylierung bei COVID-19-Patienten aufzeigt.

· Integration von Lymphom-Proteomen in eine biomedizinische Informatikplattform: Wir erstellen den grössten Proteomik-Datensatz für Lymphome mit dem Ziel, Diagnose und Behandlung zu verbessern.

· Studie an PDAC-Patienten, die eine Immuntherapie und Chemotherapie erhalten: Wir haben Patientengruppen mit prognostischen Markern für Überlebensraten identifiziert.

· Systematische Gen-Knockout-Studie in Hefe zur Untersuchung von Protein-Funktionen: Wir haben funktionelle Genomik mit Proteomik kombiniert, um Prinzipien der Protein-Funktion aufzuklären.

Molekulare Allergologie: Im Rahmen des DAViS-Zentrums wurde das GeneSelectR R-Paket erstellt und auf CRAN verfügbar gemacht. Es ermöglicht die Analyse komplexer RNA-Sequenzierungsdatensätze mit verschiedenen maschinellen Lernmethoden und die Bewertung der Klassifikationsleistung und biologischen Relevanz der Ergebnisse. Es ist Teil unserer fortlaufenden Bemühungen, molekulare und klinische Datensätze im ML-SOS-ALL-Projekt zu integrieren. Durch die Bestimmung der relativen Häufigkeit verschiedener SARS-CoV-2-Varianten im Abwasser haben wir gezeigt, dass sich eine neue Variante bei internationalen Sportveranstaltungen im Dezember 2021 und beim WEF im Januar 2023 verbreitete. Wir haben gezielte Proteomik in ex vivo differenzierten Th1-Zellen eingesetzt, um Peptide zu erkennen, die zuvor durch nicht-kodierende RNAs übersetzt wurden. In einem anderen Projekt haben wir nach Allergenproteinen in Lebensmitteln gesucht, indem wir gezielte Proteomik verwendeten.Wir haben die Proteinisoprenylierung in aktivierten Th1-Zellen weiter untersucht und die aktuelle Ansicht zu den Zielen und Orten der Proteinisoprenylierung herausgefordert. Zusätzlich zur Installation der IT-Infrastruktur gab es weitere Projekte in Zusammenarbeit zwischen dem DAViS-Zentrum und dem Zentrum für Präzisionsproteomik. Dazu gehörten ein Projekt zur verbesserten statistischen Analyse von Massenspektrometrie-Daten angereicherter Proteine sowie ein Projekt mit dem Schweizerischen Institut für Sportmedizin (SRISM) zur statistischen Analyse nicht massenspektrometrie-basierter Proteomik-Daten. Eine Veröffentlichung erfolgte in Zusammenarbeit mit der Fachhochschule Graubünden (FHGR), dem SIAF und der Medizinischen Universität Lodz in Polen, im Rahmen der gemeinsamen COVID-19-Studie. Die Studie identifizierte diagnostische Laborparameter und Prädiktoren für einen schweren Verlauf von COVID-19. Im ML-SOS-ALL-Projekt wurden Daten südafrikanischer Kinder mit und ohne atopischer Dermatitis analysiert, um relevante Transkriptlisten zu erstellen. Die Analyse von Abwasserproben und die Sequenzierung von SARS-CoV-2-RNA-Fragmenten wurden fortgesetzt und bis WEF22 abgeschlossen.

Immunmetabolismus: Das Hauptziel unserer Forschung ist es, die Wechselwirkungen zwischen immunologischen und metabolischen Signalwegen aufzuklären, um neuartige Biomarker zu identifizieren und therapeutische Zielstrukturen für die Prävention und Behandlung von respiratorischen Virusinfektionen, mikrobieller Dysbiose, allergischen Erkrankungen, Störungen der immunologischen Toleranz und unterschiedlichen Asthma-Phänotypen zu validieren. Wir analysieren patientenabgeleitete Proben aus Blut, Lunge und Haut mithilfe von Einzelzell- und Bulk-Sequenzierung, räumlicher Transkriptomik, Proteomik und Metabolomik, die mit fortschrittlichen bioinformatischen Methoden ausgewertet werden. Zur Aufklärung der zugrunde liegenden pathologischen Mechanismen setzen wir zusätzlich Geneditierung, hochdimensionale Durchflusszytometrie, konfokale Mikroskopie sowie dynamische metabolische Analysen ein, einschließlich Einzelzell-Energieprofilierung. Unsere Forschung konzentriert sich auf folgende Schwerpunkte:

· Immunologie von Virusinfektionen und deren Langzeitfolgen:
Infektionen mit Rhinoviren, respiratorischen Synzytialviren und Coronaviren verändern die angeborenen und adaptiven Immunantworten bei Patientinnen und Patienten mit chronischen Atemwegserkrankungen und Allergien. Wir haben abnormale Reaktionen auf

Bericht des Direktors

Prof. Dr. Cezmi A. Akdis

virale Infektionen in den Atemwegen sowie kurz- und langfristige immunologische Konsequenzen bei Betroffenen mit Asthma und Allergien charakterisiert.
· Immunmetabolismus bei Virusinfektionen, Immuntoleranz, Allergien und Asthma: Unsere Arbeiten umfassen (1) gestörte mitochondriale Stoffwechselprozesse in bronchialem Epithel, die zu einer verminderten antiviralen Immunantwort bei Asthma führen, (2) metabolische Kontrollpunkte für die Entwicklung humaner pathogener Th2-Zellen und regulatorischer T-Zell-Antworten, (3) immunmetabolische Umprogrammierung allergenspezifischer $CD4^+$-T-Zellen während der Allergenimmuntherapie und (4) die Effekte der Typ-2-Entzündung auf den Keratinozyten-Stoffwechsel bei atopischer Dermatitis.
· Mikrobiom und Ernährung bei allergischen Erkrankungen: Wir untersuchen, wie mikrobielle Dysbiose sowie mikrobielle und ernährungsbedingte Metabolite die Immunfunktion von Epithelzellen der Atemwege und verschiedenen T-Zell-Subtypen beeinflussen, um neue präventive Ansätze bei Asthma zu entwickeln.
· Trainierte Immunität und Toleranz als neue Ansätze für Biomarker, Prävention und Therapie bei Asthma und Allergien: Dieses Konzept beschreibt eine unspezifische immunologische Gedächtnisbildung durch epigenetische und metabolische Veränderungen. Wir analysieren diese Mechanismen in Epithelzellen der Atemwege und in T-Zellen und identifizieren Biomarker dieser neu entdeckten Prozesse im Serum von Allergiepatient*innen, insbesondere im Rahmen der Allergenimmuntherapie.

B-Zell-Immunologie: Wie aus den unten aufgeführten Projekten ersichtlich ist, ist die B-Zell-Immunologie-Gruppe hervorragend mit modernsten Techniken ausgestattet, um menschliche B-Zellen in Gesundheit und Krankheit zu untersuchen. 25 Jahre in vivo-Verfolgung von allergenspezifischen Gedächtnis-B-Zellen beim Menschen: Die Immunantwort auf Giftallergen bei Imkern stellt eines der besten Modelle dar, um die Toleranz gegenüber hohen Allergenmengen zu untersuchen. Imker und AIT-Reaktionen bei bienengiftallergischen Individuen werden im Vergleich untersucht. Charakterisierung von Gedächtnis-B-Zellen, die spezifisch für Milchallergene sind: Unsere vorläufigen Daten zeigten eine differenzielle Expression von 30 Genen, die mit der Toleranz gegenüber Lebensmittelantigenen in Zusammenhang stehen. Wir werden diese Gene mit gezielter Proteomik und B-Zell-Knock-in- und Knock-out-Experimenten eingehend analysieren. Histamin reguliert Immunantworten durch differenzielle Expression von H1- und H2-Rezeptoren: Histamin und der Histaminrezeptor (HR) 2 fördern die Entwicklung der peripheren Toleranz während der SIT und der Exposition gegenüber hohen Dosen von Gift bei Imkern. Mit diesem umfassenden Hintergrund schlagen wir vor, unsere vorläufigen Daten zur differenziellen Expression von HR1 und HR2 auf B-Zell-Untergruppen weiter zu verfolgen. Umfassende Untersuchung von Immunzellen aus asthma-diskordanten eineiigen Zwillingen: Wir untersuchen zirkulierende T- und B-Zell-Untergruppen durch umfassende Einzelzellsequenzierung kombiniert mit Einzelzell-Proteomik, um die Mechanismen der Immuntoleranz während der erdnussspezifischen OIT umfassend zu charakterisieren. Umfassende Methoden zur Untersuchung von antigenspezifischen B-Zellen werden auf Erdnussallergene und die Untersuchung von allergenspezifischen B-Zellen angewendet. Zirkulierende Lebensmittelallergen-spezifische Antikörper bei Patienten mit eosinophiler Ösophagitis: Diese Studie untersucht die zugrunde liegenden Mechanismen der eosinophilen Ösophagitis (EoE) mit Fokus auf antigenspezifische Reaktionen. Unsere Ergebnisse zeigen erhöhte Spiegel von IgG, IgG4, IgA1 und IgA2 gegen verschiedene Lebensmittelantigene bei EoE-Patienten mit signifikanten Unterschieden zwischen aktiver und inaktiver EoE. Typ 2-polarisierte Gedächtnis-B-Zellen (MBC2s): Diese Studie zeigt, dass die Dupilumab-Behandlung bei pädiatrischen Patienten mit atopischer Dermatitis (AD) die Typ 2-polarisierten Gedächtnis-B-Zellen (MBC2) und die Gesamtheit der IgE-Spiegel signifikant reduziert. Die Ergebnisse deuten darauf hin, dass MBC2- und Gesamt-IgE-Spiegel von der IL-4-Signalgebung abhängig sind und dass die Hemmung der IL-4- und IL-13-Wege durch Dupilumab AD durch die Verringerung dieser Marker effektiv behandelt. Durchflusszytometrische Analysen der Immunglobulin-Schwerketten-Isoformen des B-Zell-Rezeptors: Die genaue Erkennung der B-Zell-Rezeptor (BCR)-Schwerketten-Isoformen ist entscheidend für das Studium der B-Zell-Immunologie. In dieser Studie haben wir den Einfluss verschiedener Fc-Rezeptor (FcR)-Blockierungsreagenzien auf die Detektionsgenauigkeit der BCR-Schwerketten-Isoformen mithilfe der Durchflusszytometrie bewertet und eine optimierte Färbemethode für BCR-Isoformen entwickelt. Menschliche Askaris-Infektion und IL-10-produzierende B-Zellen: Unsere Studie ergab, dass eine Infektion mit Ascaris lumbricoides mit höheren Frequenzen von IL-10-produzierenden regulatorischen B-Zellen verbunden ist, was auf eine immunsuppressive Reaktion bei infizierten Personen hinweist. Darüber hinaus korreliert die Infektion mit reduzierten Spiegeln zirkulierender ABA-1-spezifischer IgG1- und IgE-Antikörper, was auf eine Modulation der Antikörperantworten hinweist.

Alpine Medical Campus: Das SIAF führt in enger Zusammenarbeit mit seinen unabhängigen Partnern CK-CARE, HGK, CardioCARE und der gestifteten Professur MC Brüggen auf dem Alpine Medical Campus medizinische Forschung auf höchstem Niveau durch. Diagnostik, Forschung und Therapie ergänzen sich ideal auf dem medizinischen Campus. Diese Synergien kommen den Patienten direkt zugute: Forschungsergebnisse werden in Therapieoptionen und Behandlungen umgesetzt, wodurch ein umfassendes diagnostisches und therapeutisches Konzept entsteht. Darüber hinaus sind Ausbildung, Fort- und Weiterbildung von Akademikern (MSc, PhD und Postdocs) und medizinischem Fachpersonal zentrale Bestandteile des Dienstleistungsportfolios. Das strategische Ziel des medizinischen Campus ist es, ein international anerkanntes Exzellenzzentrum im Bereich der personalisierten Prävention und Behandlung von allergischen und kardiovaskulären Erkrankungen zu schaffen. Wir freuen uns sehr auf eine umfangreiche Zusammenarbeit im Rahmen des neu entstehenden Alpine Innovationszentrums.

Schweizerisches Forschungsinstitut für Sportmedizin: Nach der Gründung des Schweizerischen Forschungsinstituts für Sportmedizin (SRISM) hat sich unsere Forschungszusammenarbeit effizient weiterentwickelt und konzentriert sich auf Eishockeyspieler, Langläufer, nicht-professionelle Athleten und nicht sportliche Kontrollen. Wir untersuchten die Mechanismen häufiger Atemwegsinfektionen und Infektionskrankheiten bei Athleten sowie die immunologischen Mechanismen des Trainings. Wir erreichten

Prof. Dr. Cezmi A. Akdis

die Veröffentlichung des 5. gemeinsamen Artikels und mehrere Schlüsselstudien sind im Gange, die sich auf die Auswirkungen des Trainings, des Immunstoffwechsels, des Geschlechts, der Auswirkungen der Umweltbelastung, der körperlichen Leistungsfähigkeit und Leistung konzentrieren.

Ausbildung, Lehre, Kongresse: Das SIAF spielt eine bedeutende Rolle in der Ausbildung von Studierenden sowie in der postgradualen Weiterbildung. Gleichzeitig nimmt das Institut Lehrverpflichtungen an der Universität Zürich wahr. Diese umfassen unter anderem Vorlesungen im Fach Biochemie am Biochemischen Institut.
Prof. C. A. Akdis ist Mitglied der Medizinischen Fakultät der Universität Zürich und besitzt das Promotionsrecht an der Mathematisch-Naturwissenschaftlichen Fakultät. Zudem ist er Ehrenprofessor an der Bezmialem Universität in Istanbul. Darüber hinaus halten Prof. C. A. Akdis und Prof. M. Akdis Ehrenprofessuren an mehreren renommierten Institutionen, darunter das Tongren Hospital der Peking University, die Universität Bursa-Uludağ, die Wuhan University, das T.H. Chan Institute of Epidemiology der Harvard University sowie die Zhejiang University in China. Auch Prof. Christoph Messner, PD Dr. K. Bärenfaller und PD Dr. M. Sokolowska gehören zum Lehrkörper der Universität Zürich. PD Dr. K. Bärenfaller besitzt das Promotionsrecht an der Mathematisch-Naturwissenschaftlichen Fakultät der Universität Zürich.

Auszeichnungen: Prof. Dr. C.A. Akdis 2024 wurde zum neunten Mal von Thomson Reuters Clarivate in die Liste der meistzitierten Forscher aus allen wissenschaftlichen Disziplinen weltweit aufgenommen. Milena Sokolowska gehört zum zweiten Mal zu Stanford/Elsevier's 2024 Top 2% Wissenschaftlerinnen und erstmals zu den am meisten zitierten Wissenschaftlern (1% Highly Cited Researcher 2024 Clarivate).

Klinischer Dienst: Seit 2019 ist das SIAF Dienstleister der OLINK-Technologie und bietet seinen akademischen und industriellen Kunden spezielle Messungen zur Bestimmung von Biomarkern an, die als Parameter zur Beurteilung der Krankheitsentwicklung und -überwachung dienen. Diese Messungen helfen auch, verschiedene Manifestationen und Verläufe von Krankheiten besser zu verstehen. Derzeit ist das SIAF das einzige Labor in der Schweiz, das diese speziellen Messungen sowohl für nationale als auch für internationale Zwecke anbietet. Das SIAF stellt der Davoser Klinik und allen anderen interessierten Kliniken und praktizierenden Ärzten spezielle zelluläre immunologische Untersuchungen zur Verfügung. Mithilfe der Durchflusszytometrie (FACS-Analyse) von Blut, bronchoalveolärer Lavage (BAL) sowie anderen Gewebeflüssigkeiten werden die verschiedenen Immunzellen und Unterpopulationen in Bezug auf ihre Entwicklung, Anteile und Aktivierungszustand gemessen.

Biomedical Data Mining Block Course (BME351): Die fünfte Durchführung des Blockkurses „Biomedical Data Mining“ an der Universität Zürich fand im Frühjahrssemester vom 4. April bis 7. Mai 2024 statt. Der Kurs begann in Präsenz am SIAF mit einer ersten Einheit am Freitag, gefolgt von Nachmittags- und Ganztagessitzungen von Dienstag bis Freitag in der darauffolgenden Woche und wurde anschließend online fortgeführt. Die inhaltliche Leitung lag bei PD Dr. K. Bärenfaller, PD Dr. M. Sokolowska und Prof. Dr. Christoph Messner. Im Rahmen des Kurses erhielten die Teilnehmenden Datensätze aus verschiedenen RNA-Sequenzierungs- oder Proteomik-Projekten. Ziel war es, diese Daten mit Hilfe der Programmiersprache R zu analysieren, Ergebnisse zu interpretieren und in einen wissenschaftlichen Kontext einzuordnen. Im Sinne forschungsorientierten Lernens wurden die Studierenden zudem ermutigt, eigene Fragestellungen auf Basis der bereitgestellten Datensätze zu entwickeln. Zur Vermittlung der notwendigen fachlichen und methodischen Grundlagen wurde der praktische Teil durch Vorlesungen zu Themen wie Proteomik, Transkriptomik, Metabolomik, Durchflusszytometrie, Versuchsdesign und statistischen Methoden, funktioneller Kategorisierung, maschinellem Lernen sowie KI-basierten Analysewerkzeugen ergänzt. Darüber hinaus erhielten die Teilnehmenden Einblicke in laufende Promotionsprojekte der betreuenden Doktorierenden. Zum Abschluss des Kurses präsentierten die Studierenden ihre Ergebnisse im Rahmen eines mündlichen Vortrags und reichten einen schriftlichen Bericht zu ihrer Data-Mining-Aufgabe ein.

Mechanismen des Verlaufs allergischer Erkrankungen (BME364). Die erste Durchführung dieses Blockkurses an der Universität Zürich fand im Herbstsemester 2024 vom 10. bis 31. Oktober 2024 statt. Verantwortliche Dozierende waren Dr. Willem van de Veen, PD Dr. M. Sokolowska, Dr. Yasutaka Mitamura und Dr. Ismail Ogulur. Der Kurs beleuchtete die immunologischen und molekularen Mechanismen, die allergischen Erkrankungen zugrunde liegen. Die Studierenden beschäftigten sich mit der Pathophysiologie, Diagnostik und Therapie von Erkrankungen wie Asthma, Nahrungsmittelallergien und atopischer Dermatitis. Durch eine Kombination aus Vorlesungen, praktischen Laborübungen vor Ort in Davos sowie Fallstudien erhielten die Teilnehmenden ein umfassendes Verständnis sowohl der theoretischen Grundlagen als auch der klinischen Aspekte allergischer Erkrankungen. Die Auseinandersetzung mit aktueller Forschung, modernen Diagnoseverfahren und innovativen Therapien vermittelte den Studierenden die notwendigen Kenntnisse, um zur Weiterentwicklung der Allergiebehandlung und -prävention beizutragen.

Workshop „Grundlagen der Hochschuldidaktik am SIAF“: Gefördert durch die ULF – Subvention für hochschuldidaktische Grundlagenausbildung – fand im November 2024 am SIAF ein zweitägiger Workshop zum Thema „Grundlagen der Hochschullehre und -didaktik am SIAF“ statt. Neun Mitarbeitende des SIAF nahmen daran teil. Der Workshop wurde von Dr. Svenja Kaduk und Anja Pawelleck von der Weiterbildung der Universität Zürich geleitet. Die Teilnehmenden erhielten praxisnahe Schulungen zu Themen wie Kursplanung, Aktivierung von Studierenden, konstruktives Feedback sowie Betreuungsprozesse in der Lehre.

Weltkonferenz zur Immunregulation: Nach den Jahren der Pandemie konnte die 19. Ausgabe der Weltkonferenz zur Immunregulation (WIRM) zum dritten Mal in Folge wieder als Präsenzveranstaltung im Kongresszentrum Davos stattfinden. Rund 350 Wissenschaftler aus über 25 Ländern der Welt versammelten sich vom 13. – 16. März 2024 zu dem viertägigen Kongress, um die neuesten Erkenntnisse in der Immunologie auszutauschen. Talentierte junge

Bericht des Direktors

Prof. Dr. Cezmi A. Akdis

Forscher trafen auf erfahrene Experten, als Wissenschaftler 119 Vorträge hielten und 180 Abstracts präsentierten, in denen sie ihre neuesten Erkenntnisse in der Immunologie teilten. Dieser globale Austausch aktueller Erkenntnisse hilft, neue Therapieansätze und innovative Behandlungsmethoden für Patienten zu entwickeln.

Finanzielle Basis: Die Ausgaben und Einnahmen des SIAF haben sich im Vergleich zu den Vorjahren nur geringfügig verändert. Die Grundfinanzierung des Instituts wird derzeit durch die Hauptsponsoren sichergestellt. Sie besteht hauptsächlich aus einem Beitrag des Bundes (Forschungsgesetz Art. 15), Beiträgen des Kantons Graubünden und der Gemeinde Davos, Beiträgen der Universität Zürich, Beiträgen des Schweizerischen Nationalfonds, EU-Konsortialzuschüssen Syn-Air-G sowie Beiträgen von Stiftungen wie der PROMEDICA Stiftung und der Stiftung vormals Bündner Heilstätte Arosa, die Doktorandenprogramme unterstützen. Die zusätzlichen Ausgaben wurden durch Einnahmen aus zusätzlich wettbewerblich erworbenen Drittmitteln und dem WIRM-Kongress gedeckt.

Danksagungen:
Ich möchte allen Mitarbeitern herzlich für ihre grossartige Arbeit und das gute Arbeitsklima am SIAF danken. Gleichzeitig danke ich den Davoser Kliniken, ihren Chefärzten und ihrem Personal sowie der Universität Zürich für ihre konstante und effektive Unterstützung unseres Instituts.

Besonders hervorheben möchte ich unsere fruchtbare Zusammenarbeit mit CK-CARE, die es uns ermöglicht, patientenorientierte Forschung bei atopischer Dermatitis durchzuführen. Ich danke insbesondere Frau und Herrn Kühne für ihre Unterstützung, die unsere Forschung befähigt, nachhaltige Lösungen für bessere Diagnosen und Behandlungen von Patienten mit atopischer Dermatitis zu finden. Dank dieser Unterstützung wurden im Institut viele Master- und Doktorgrade erworben und eine grosse Anzahl internationaler Stipendien vergeben, ein sehr erfolgreiches Projekt, das seit der Gründung von CK-CARE über 60 kurzfristige internationale Stipendien unterstützt hat.

Mein ausserordentlicher Dank gilt auch der Stiftung Schweizerisches Forschungsinstitut für Hochgebirgsklima und Medizin (SFI), ihrem Stiftungsrat und ihrem Ausschuss für die stets gewährte Unterstützung. Mein Dank und meine besten Wünsche für viel Erfolg gehen an Brigitta Gadient, die neue Präsidentin unserer Stiftung. Last but not least, mein besonderer Dank geht an den Kanton Graubünden, das Staatssekretariat für Bildung, Forschung und Innovation und alle Behörden, die ein entschlossenes Interesse an der Forschung des SIAF gezeigt haben und das Institut kontinuierlich unterstützen.

Davos, Mai 2025

SIAF, CK-Care and HGK members during sports night

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. The goal is to develop treatments that will improve the future of those affected. 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 and asthmatic diseases. 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.

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 2024, 78 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 **906.094 and an average of 11.617 points** per publication in 2024. The latest findings were also presented in **70 abstracts** at various professional conferences. Our employees were invited to participate in **123 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 **37** 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 12.6** 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 ninth time in 2024. In 2024, PD Dr. Milena Sokolowska, a Head of SIAF Immune Metabolism Group, has been also listed at this list for the first time. The SIAF has published around 1,783 scientific contributions and is among the most cited institutes worldwide. The articles published by the SIAF have been **cited 97'000 times**.

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, mi-

Report of the director

Prof. Dr. Cezmi A. Akdis

crobiome 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.

Precision Proteomics Center: One of the key projects of the Institute is the establishment of the Precision Proteomics Center at SIAF in collaboration with the Canton of Grisons and the University of Zurich. The center was successfully established and is now in the process of staffing and infrastructure expansion under the direction of Prof. Christoph Messner. The center develops and applies state-of-the-art mass spectrometry-based technologies for proteome analysis of clinical samples. The goal of the center is to identify novel biomarkers and disease mechanisms that will contribute to the development of the next generation of personalized treatments.
· Established high-throughput proteomics technologies for applications in biomarker discovery studies for allergies, skin diseases, and oncology.
· New technique 'OxoScan-MS' for large-scale plasma glycoproteomics: We developed a method that enables quantitative mapping of glycopeptides in large cohorts, revealing differential glycosylation in COVID-19 patients.
· Integrating lymphoma proteomes into a biomedical informatics platform: We are generating the largest proteomics dataset for lymphoma, aiming to improve diagnosis and treatment.
· Study on PDAC patients receiving immunotherapy and chemotherapy: We identified patient subgroups with prognostic markers for survival outcomes.
· Systematic gene knockout in yeast to study protein functions: We combined functional genomics with proteomics to reveal principles of protein.

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 subsets T cells 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.

Prof. Dr. Cezmi A. Akdis

B cell Immunology: As can be seen from the below projects, the B cell immunology group has been extremely well equipped with front techniqes to investigate human B cells in health and diseases. 25 years 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. Bee keepers and AIT response in bee venom allergic individuals will be investigated in comparison. Characterization of memory B cells specific for milk allergens: Our preliminary data demonstrated differential expression 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 promotes 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.To comprehensively characterize the mechanisms of immune tolerance during the peanut-specific OIT. Extensive methodology to investigate antigen-specific B cells is goingto be applied to peanut allergens and the investigation of allergen-specific B cells. 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 and total IgE levels are dependent on IL-4 signaling, with dupilumab's inhibition of IL-4 and IL-13 pathways effectively managing AD by decreasing 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. In this study, we evaluated the impact of various
Fc receptor (FcR) blocking reagents on the detection accuracy of BCR heavy chain isotypes using flow cytometry and developed an optimized staining method for BCR isotypes. 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, suggesting an immunosuppressive response in infected individuals. 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, Cardio-CARE and the and the endowed professorship MC Brüggen on the Alpine Medical Campus, conducts cutting-edge medical research. Diagnostics, research, and therapy complement each other ideally on the medical campus. These synergies directly benefit the patients: research findings are translated into therapy options and treatments, allowing for a comprehensive diagnostic and therapeutic concept. Furthermore, education, training, and continuing education of academics (MSc, PhD, and postdocs) and medical professionals are central components of the service portfolio. The strategic goal of the medical campus is to create an internationally recognized center of excellence in the field of personalized prevention and treatment of allergic and cardiovascular diseases. We very much look forward to extensive collaboration in the framework of the newly developing Alpine Innnovation 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 athlethes 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 permance.

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 2024, Prof. Dr. C.A. Akdis was named for the ninth time to the list of the world's most highly cited researchers across all scientific disciplines by Thomson Reuters Clarivate. Milena Sokolowska was included for the second time in Stanford/Elsevier's 2024 list of the top 2% of scientists worldwide and was also recognized for the first time as a 2024 Highly Cited Researcher (top 1%) by Clarivate.

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 cy-

Report of the director

Prof. Dr. Cezmi A. Akdis

tometric 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.

Biomedical Data Mining Block Course (BME 351): The fifth edition of the block course "Biomedical Data Mining" at the University of Zurich took place during the spring semester, from 4 April to 7 May 2024. The course was initially held on-site at SIAF, starting on the first Friday and continuing from Tuesday afternoon to Friday in the following week, and was then continued online. The responsible lecturers were PD Dr K. Bärenfaller, PD Dr M. Sokolowska, and Prof. Dr Christoph Messner. During the course, students were given data from various RNA sequencing or proteomics projects and were asked to analyse these datasets using R across a range of tasks, followed by contextualising the results. Applying the concept of research-based learning, students were also encouraged to develop their own research questions using the data. To equip students with the necessary skills and background, the hands-on component was supplemented with lectures on proteomics, transcriptomics, metabolomics, flow cytometry, experimental design and statistical methods, functional categorisation, machine learning, and AI tools. In addition, they were introduced to the research projects of the PhD students supervising the course. At the end of the course, students were required to give an oral presentation and submit a written report on their data mining task.

Mechanisms of allergic disease course (BME364). The first edition of this block course at the University of Zurich took place during the fall semester 2024 from 10th till 31st October 2024. The responsible lecturers were Dr. Willem van de Veen, PD Dr. M. Sokolowska, Dr. Yasutaka Mitamura and Dr. Ismail Ogulur. This course explored the immunological and molecular mechanisms underlying allergic diseases. Students studied the pathophysiology, diagnosis, and treatment of conditions such as asthma, food allergies, and atopic dermatitis. The course combined lectures, practical laboratory hands-in sessions on-site in Davos, and case studies to provide a comprehensive understanding of both theoretical and clinical aspects of allergy. By examining current research, diagnostic methods, and emerging therapies, students gained the skills needed to contribute to advancements in allergy treatment and management.

Supported by the ULF – Subsidy for Foundational University Didactics Training, the two-day workshop "**Basics of University Teaching and Learning at SIAF**" was held at SIAF in November 2024, with the participation of nine SIAF employees. The workshop was led by Dr. Svenja Kaduk and Anja Pawelleck from UZH Continuing Education, who provided training on course planning, student engagement, constructive feedback, and supervision processes.

19th World Immune Regulation Meeting: After years of the pandemic, the 19th edition of the World Immune Regulation Meetings (WIRM) could finally be held for the second time in a row as a face-to-face meeting at the Davos Congress Center. Around 350 scientists from over 25 countries around the world gathered from March 13 – 16, 2024, for the four-day congress to exchange the latest findings in immunology. Talented young researchers met experienced experts, as scientists delivered 119 presentations and presented 180 abstracts, sharing their latest insights in immunology. This global exchange of current knowledge helps develop new treatment therapies and innovative approaches for patients.

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

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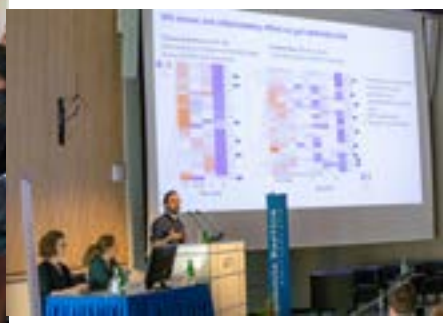
My deep appreciation also goes to the Swiss Research Institute for High Altitude Climate and Medicine Foundation (SFI), its Board of Trustees, and its Executive Committee for their unwavering support. I would like to extend my sincere thanks and best wishes for continued success to Brigitta Gadient,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

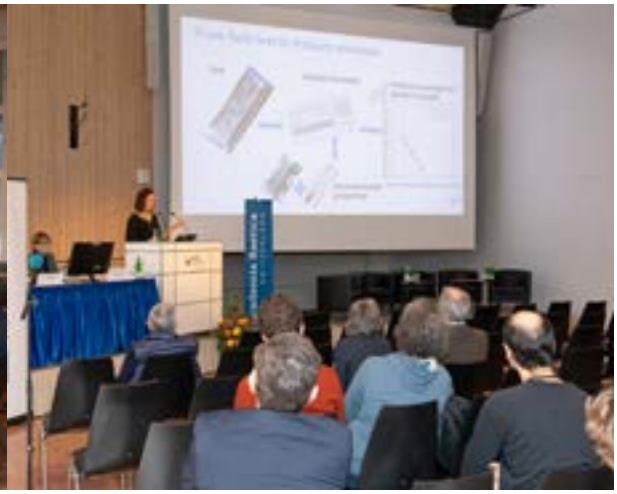
Davos, May 2025

Prof. Dr. Cezmi A. Akdis



SIAF @ Graubünden forscht

November 2024



2024

Prof. Dr. Cezmi A. Akdis

Director

Akdis Cezmi A.	Prof., MD ***

Group Leaders

Akdis Cezmi A.	Prof., MD, Head Immunology ***
Akdis Mübeccel	Prof, MD, PhD, Head Immune Regulation *
Bärenfaller Katja	PD, PhD, Head Molecular Allergology *
Messner Christoph	Prof., PhD, Head Precision Proteomics ***
Rhyner Claudio	PhD, Head Vaccine Development *
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The epithelial barrier theory: A key player in health and chronic non-communicable diseases

Main Contributors of the Onset, Exacerbation and Severity of Chronic Non-Communicable Diseases

Exposure to air pollution, processed foods, cleaning products, microplastics and environmental toxins represents a major culprit of the onset, exacerbation and severity of chronic non-communicable diseases as explained by the epithelial barrier theory.

In addition, an unhealthy diet, particularly high sugar intake and other carbohydrates, and general high caloric intake leading to obesity are linked to metabolic dysregulation and epithelial barrier leakiness and have been implicated in many chronic non-communicable diseases. Poor physical activity and excessive alcohol consumption can further aggravate these factors. Poverty and social inequalities in access to healthcare, healthy food, and education, urbanization, and modern food diets characterized by processed foods, sedentary lifestyles, further contribute to the development of many chronic non-communicable diseases.

Does the Epithelial Barrier Theory Explain the Rise in Non-communicable Diseases?

Yes, 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.

What are the Most Harmful Environmental Agents that Damage the Epithelial Barriers?

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, 80), preservatives, enzymes; micro- and nanoplastics; cigarette smoke; chlorine in water and swimming pools and pesticides.

What are the Key Physiopathological Findings Presented by the Epithelial Barrier Theory?

Epithelial Barrier Disruption Triggers Disease: Environmental agents damage epithelial barriers, increasing the permeability and facilitating the entry of allergens, toxins, and microbes into the tissues.

Chronic Inflammation (“Epithelitis”): Epithelial barrier damage initiates danger signals, release of alarmins, multiple proinflammatory cytokines and chemokines and causes persistent inflammation.

Microbial Imbalance (Dysbiosis): Epithelial barrier injury and an inflammatory immune response against microbes is characterized by a loss of commensals and colonization of opportunistic pathogens.

Rise in noncommunicable diseases (NCDs): The increase in allergic, autoimmune, metabolic, and neuropsychiatric diseases since the 1960s is linked to the widespread exposure to toxic substances.

Shared Molecular Mechanisms Across Various Diseases: Certain chronic diseases share pathogenic features such as epithelial barrier dysfunction, microbial imbalance, oxidative stress, mitochondrial and endoplasmic reticulum stress, DNA repair errors, and chronic inflammation.

Integration of Previous Hypotheses: The epithelial barrier theory embodies older ideas, including the “hygiene hypothesis” and “biodiversity hypothesis.”

The journey of our discoveries leading to the epithelial barrier theory

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. 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. 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 non-communicable 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, where they serve as the primary line of defense due to the absence of a protective stratum corneum.

Around 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. Our research, which centered on the skin, led us to further validate our findings using human organoids and organs-on-a-chip. Currently, we are conducting studies in human native skin explants using advanced skin culture methods. Through this research, we identified various harmful substances that humans have been inadvertently exposed to since the 1960s. The introduction of sodium lauryl sulfate (SLS or SDS: sodium dodecyl sulfate) into detergents and other cleaning products represents an example of innovative products developed during that time using additives that are now demonstrated to have an impact on our health. The trend continues with the addition of enzymes to detergents, such as amylase, lipase, and protease, that impair the epithelial barrier function. Exposure to other factors, such as air pollution and food additives present in our modern diets, further compromises the epithelial barriers.

These harmful substances that have become more prevalent in our daily lives due to industrialization and modernization can impair the protective barriers in the skin, respiratory system, and gastrointestinal tract. This disruption can lead to imbalances in the microbial community and shift it towards a proinflammatory state. Examples of substances that can damage these barriers include surfactants and enzymes found in cleaning products, as well as chlorine in swimming pools, microplastics, and air pollutants such as ozone, particulate matter, and diesel exhaust. Our systematic research of epithelial barriers spanning across 26 years led to the extended “epithelial barrier theory”, which attributes the rise of allergic and noncommunicable inflammatory diseases to our increased exposure to toxic agents.

Which Diseases are Triggered by Epithelial Barrier Dysfunction and Microbial Dysbiosis?

The epithelial barrier theory and its associated diseases.
Sun N, Ogulur I, Mitamura Y, Yazici D, Pat Y, Bu X, Li M, Zhu X, Babayev H, Ardicli S, Ardicli O, D'Avino P, Kiykim A, Sokolowska M, van de Veen W, Weidmann L, Akdis D, Ozdemir BG, Brüggen MC, Biedermann L, Straumann A, Kreienbühl A, Guttman-Yassky E, Santos AF, Del Giacco S, Traidl-Hoffmann C, Jackson DJ, Wang DY, Lauerma A, Breiteneder H, Zhang L, O'Mahony L, Pfaar O, O'Hehir R, Eiwegger T, Fokkens WJ, Cabanillas B, Ozdemir C, Kistler W, Bayik M, Nadeau KC, Torres MJ, Akdis M, Jutel M, Agache I, Akdis CA. Allergy. 2024 Dec;79(12):3192-3237. doi: 10.1111/all.16318.

We have recently reported that the pathogenesis of 68 major diseases is linked to the epithelial barrier theory. In recent years, the epithelial barrier theory has come to light, explaining the growing prevalence and exacerbations of these diseases worldwide. It attributes their onset to a functionally impaired epithelial barrier triggered by the toxicity of the exposed substances, associated with microbial dysbiosis, immune system activation, and inflammation. Diseases encompassed by the epithelial barrier theory share common features such as an increased prevalence after the 1960s or 2000s that cannot (solely) be accounted for by the emergence of improved diagnostic methods. Other common traits include epithelial barrier defects, microbial dysbiosis with loss of commensals and colonization of opportunistic pathogens, and circulating inflammatory cells and cytokines. In addition, practically unrelated diseases that fulfill these criteria have started to emerge as multimorbidities during the last decades. Here, we provide a comprehensive overview of diseases encompassed by the epithelial barrier theory and discuss studies supporting their shared epidemiology, genetic susceptibility, epithelial barrier dysfunction, microbial dysbiosis, and tissue inflammation.

Implications of the Epithelial Barrier Theory on the Health and Performance of Elite Athletes

Epithelial barrier theory in the context of nutrition and environmental exposure in athletes.
Kistler W, Villiger M, Villiger B, Yazici D, Pat Y, Mitamura Y, Ardicli S, Skolnick S, Dhir R, Akdis M, Nadeau K, Ogulur I, Akdis CA. Allergy. 2024 Nov;79(11):2912-2923. doi: 10.1111/all.16221.

Exposure to toxic substances, introduced into our daily lives during industrialization and modernization, can disrupt the epithelial barriers in the skin, respiratory, and gastrointestinal systems, leading to microbial dysbiosis and inflammation. Athletes and physically active individuals are at an increased risk of exposure to agents that damage the epithelial barriers and microbiome. Their extreme physical exercise exerts stress on many organs, resulting in tissue damage and inflammation. Epithelial barrier-damaging substances include surfactants and enzymes in cleaning products, laundry and

dishwasher detergents, chlorine in swimming pools, microplastics, air pollutants such as ozone, particulate matter, and diesel exhaust. 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.

One Health: The Exposure of Pets and Domestic Animals to Epithelial Barrier-Damaging Substances In the Same Way as Humans and Their Diseases

Epithelial barrier dysfunction and associated diseases in companion animals: Differences and similarities between humans and animals and research needs.

Ardicli S, Ardicli O, Yazici D, Pat Y, Babayev H, Xiong P, Zeyneloglu C, Garcia-Sanchez A, Shi LL, Viscardi OG, Skolnick S, Ogulur I, Dhir R, Jutel M, Agache I, Janda J, Pali-Schöll I, Nadeau KC, Akdis M, Akdis CA. Allergy. 2024 Dec;79(12):3238-3268. doi: 10.1111/all.16343.

Since the 1960s, more than 350,000 new chemicals have been introduced into the lives of humans and domestic animals. Many of them have become part of modern life and some are affecting nature as environmental pollutants. Yet, there are limited studies of their potential health risks for both humans and animals in the context of the epithelial barrier. Impaired epithelial barriers, microbial dysbiosis, and tissue inflammation have been observed in a high number of mucosal inflammatory, autoimmune and neuropsychiatric diseases, many of which are increasingly common. 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. In this review, we explore the implications of damaged epithelial barriers on the well-being of companion animals, drawing comparisons with humans, and aim to elucidate the spectrum of diseases that afflict them. In addition, future research areas focusing on the interconnectedness of human, animal, and environmental well-being are highlighted in line with the "One Health" concept.

EAACI guidelines on environmental science for allergic diseases and asthma

The impact of indoor pollution on asthma-related outcomes: A systematic review for the EAACI guidelines on environmental science for allergic diseases and asthma.

Agache I, Canelo-Aybar C, Annesi-Maesano I, Cecchi L, Biagioni B, Chung F, D'Amato G, Damialis A, Del Giacco S, De Las Vecillas L, Dominguez-Ortega J, Galàn C, Gilles S, Giovannini M, Holgate S, Jeebhay M, Nadeau K, Papadopoulos N, Quirce S, Sastre J, Traidl-Hoffmann C, Walusiak-Skorupa J, Sousa-Pinto B, Salazar J, Rodríguez-Tanta LY, Cantero Y, Montesinos-Guevara C, Song Y, Alvarado-Gamarra G, Sola I, Alonso-Coello P, Nieto-Gutierrez W, Jutel M, Akdis CA. Allergy. 2024 Jul;79(7):1761-1788. doi: 10.1111/all.16051.

Systematic review following the GRADE approach on the impact of exposure to volatile organic compounds (VOCs), cleaning agents, mould/damp, pesticides on the risk of (i) new-onset asthma (incidence) and (ii) adverse asthma-related outcomes (impact). MEDLINE, EMBASE and Web of Science were searched for indoor pollutant exposure studies reporting on new-onset asthma and critical asthma-related outcomes. Ninety-four studies were included: 11 for VOCs (7 for incidence and 4 for impact), 25 for cleaning agents (7 for incidence and 8 for impact), 48 for damp/mould (26 for incidence and 22 for impact) and 10 for pesticides (8 for incidence and 2 for impact). Exposure to damp/mould increases the risk of new-onset wheeze (moderate certainty evidence). Exposure to cleaning agents may be associated with a higher risk of new-onset asthma and with asthma severity (low level of certainty). Exposure to pesticides and VOCs may increase the risk of new-onset asthma (very low certainty evidence). The impact on asthma-related outcomes of all major indoor pollutants is uncertain. As the level of certainty is low or very low for most of the available evidence on the impact of indoor pollutants on asthma-related outcomes, more rigorous research is warranted.

EAACI guidelines on environmental science for allergy and asthma: The impact of short-term exposure to outdoor air pollutants on asthma-related outcomes and recommendations for mitigation measures.

Agache I, Annesi-Maesano I, Cecchi L, Biagioni B, Chung KF, Clot B, D'Amato G, Damialis A, Del Giacco S, Dominguez-Ortega J, Galàn C, Gilles S, Holgate S, Jeebhay M, Kazadzis S, Nadeau K, Papadopoulos N, Quirce S, Sastre J, Tummon F, Traidl-Hoffmann C, Walusiak-Skorupa J, Jutel M, Akdis CA. Allergy. 2024 Jul;79(7):1656-1686. doi: 10.1111/all.16103.

The EAACI Guidelines on the impact of short-term exposure to outdoor pollutants on asthma-related outcomes provide recommendations for prevention, patient care and mitigation in a framework supporting rational decisions for healthcare professionals and patients to personalize and improve asthma management. Moreover, it provides policymakers and regulators with science-based evidence to guide the implementation of legally binding standards and goals for outdoor air quality at international, national and local levels. The Guideline was developed using the GRADE approach and evaluated outdoor pollutants referenced in the current Air Quality Guideline of the World Health Organization as single or mixed pollutants and outdoor pesticides. Short-term exposure to all pollutants evaluated increased the risk of asthma-related adverse outcomes, especially hospital admissions and emergency department visits (moderate certainty of evidence at specific lag days). There is limited evidence of the impact of traffic-related air pollution and outdoor pesticide

exposure and the interventions implemented to reduce emissions. Given the quality of evidence, conditional recommendations were formulated for all pollutants and the interventions for mitigating outdoor air pollution. Asthma management counselled by the current EAACI guidelines can improve asthma-related outcomes but global measures for clean air are needed to achieve a significant impact.

The impact of exposure to tobacco smoke and e-cigarettes on asthma-related outcomes: Systematic review informing the EAACI guidelines on environmental science for allergic diseases and asthma.

Agache I, Ricci-Cabello I, Canelo-Aybar C, Annesi-Maesano I, Cecchi L, Biagioni B, Chung KF, D'Amato G, Damialis A, Del Giacco S, De Las Vecillas L, Dominguez-Ortega J, Galán C, Gilles S, Giovannini M, Holgate S, Jeebhay M, Nadeau K, Papadopoulos N, Quirce S, Sastre J, Traidl-Hoffmann C, Walusiak-Skorupa J, Salazar J, Sousa-Pinto B, Colom M, Fiol-deRoque MA, Gorreto López L, Malih N, Moro L, Pardo MG, Pazo PG, Campos RZ, Saletti-Cuesta L, Akdis M, Alonso-Coello P, Jutel M, Akdis CA. Allergy. 2024 Sep;79(9):2346-2365. doi: 10.1111/all.16151.

A systematic review (SR) was performed by the European Academy of Allergy and Clinical Immunology using the GRADE approach to develop the clinical practice guidelines on the impact of environmental tobacco smoke (ETS) and active smoking on the risk of new-onset asthma/recurrent wheezing (RW)/low lung function (LF), and on asthma-related outcomes. Only longitudinal studies were included, almost all on combustion cigarettes, only one assessing e-cigarettes and LF. According to the first SR (67 studies), prenatal ETS increases the risk of RW (moderate certainty evidence) and may increase the risk of new-onset asthma and of low LF (low certainty evidence). Postnatal ETS increases the risk of new-onset asthma and of RW (moderate certainty evidence) and may impact LF (low certainty evidence). Combined, in utero and postnatal ETS, may increase the risk of new-onset asthma (low certainty evidence) and increase the risk of RW (moderate certainty evidence). The second SR (24 studies) concluded that ETS increases the risk of severe asthma exacerbations and impairs asthma control and LF (moderate certainty evidence). According to the third SR (25 studies), active smoking increases the risk of severe asthma exacerbations and of suboptimal asthma control (moderate certainty evidence) and may impact asthma-related quality-of-life and LF (low certainty evidence).

The impact of outdoor pollution and extreme temperatures on asthma-related outcomes: A systematic review for the EAACI guidelines on environmental science for allergic diseases and asthma

Agache I, Canelo-Aybar C, Annesi-Maesano I, Cecchi L, Rigau D, Rodríguez-Tanta LY, Nieto-Gutierrez W, Song Y, Cantero-Fortiz Y, Roqué M, Vasquez JC, Sola I, Biagioni B, Chung F, D'Amato G, Damialis A, Del Giacco S, Vecillas LL, Dominguez-Ortega J, Galàn C, Gilles S, Giovannini M, Holgate S, Jeebhay M, Nadeau K, Papadopoulos N, Quirce S, Sastre J, Traidl-Hoffmann C, Walusiak-Skorupa J, Sousa-Pinto B, Alonso-Coello P, Salazar J, Jutel M, Akdis CA. Allergy. 2024 Jul;79(7):1725-1760. doi: 10.1111/all.16041.

Air pollution is one of the biggest environmental threats to asthma, which is aggravated by climate change. To inform the recommendations of the EAACI Guidelines on the environmental science for allergic diseases and asthma, a systematic review (SR) evaluated the impact on asthma-related outcomes of short-term exposure to outdoor air pollutants (PM2.5, PM10, NO2, SO2, O3, and CO), heavy traffic, outdoor pesticides, and extreme temperatures. Additionally, the SR evaluated the impact of governmental regulatory policies to reduce outdoor pollutants on asthma-related outcomes. The risk of bias was assessed using ROBINS-E tools and the certainty of the evidence using the GRADE approach. Short-term exposure to PM2.5, PM10, and NO2 has been associated with an increased risk of asthma-related hospital admissions (HA) and emergency department (ED) visits (moderate certainty evidence). Exposure to heavy traffic may increase HA and deteriorate asthma control (low certainty evidence). Interventions reducing outdoor pollutants may reduce asthma exacerbations (low to very low certainty evidence). Exposure to fumigants may increase the risk of new-onset asthma in agricultural workers, while exposure to 1,3-dichloropropene may increase the risk of asthma-related ED visits (low certainty evidence). Abrupt temperature changes may increase the risk of asthma-related ED visits and HA and asthma mortality (low certainty evidence).

Monkeypox 2024 outbreak: Fifty essential questions and answers

Cabanillas B, Murdaca G, Guemari A, Torres MJ, Azkur AK, Aksoy E, Vitte J, Fernández-Santamaria R, Karavelia A, Castagnoli R, Valdelvira R, Orsi A, Ogliastro M, Massaro E, Yücel EÖ, Novak N, Agache I, Akdis M, Akdis CA. Allergy. 2024 Dec;79(12):3285-3309. doi: 10.1111/all.16374.

As the world still vividly recalls the previous monkeypox (mpox) outbreak that impacted over 120 countries worldwide with more than 99,000 cases in 2022, we are now facing a second wave of infections from the monkeypox virus (MPXV). The current 2024 outbreak has already recorded more than 20,000 cases in Africa, marking a dramatic escalation compared to previous outbreaks. The predominance of the newly identified clade Ib variant, first detected in the Democratic Republic of the Congo (DRC) and now identified across multiple African nations and beyond, underscores its enhanced transmissibility and potential for international spread, evidenced by cases in Sweden and Thailand. The World Health Organization (WHO) declared on August 14, 2024, the current mpox outbreak a Public Health Emergency of International Concern (PHEIC), calling for heightened global public health measures. The ongoing pattern of unusual, frequent, and extensive outbreaks of mpox with potential global implications poses significant questions. This review addresses, in the format of 50 questions and answers, the 2024 mpox outbreak, detailing its characteristics, epidemiological data, and impact compared to previous outbreaks. It comprehensively explores critical questions related to MPXV virological characteristics, immunological response, clinical manifestations, epidemiology, diagnostics, and available treatments. The review also documents the significant and evolving challenges posed by the current mpox outbreak, highlighting its scale, spread, and public health response.

Epithelial barrier dysfunction, type 2 immune response, and the development of chronic inflammatory diseases

Ogulur I, Pat Y, Yazici D, Ardicli Ardicli SO, Mitamura Y, Akdis M, Akdis CA. Curr Opin Immunol. 2024 Dec;91:102493. doi: 10.1016/j.coi.2024.102493

The prevalence of many chronic noncommunicable diseases has been steadily rising over the past six decades. During this time, humans have been increasingly exposed to chemicals that damage epithelial cells, including air pollutants, laundry and dishwashers, household chemicals, toothpaste, food additives, microplastics, and nanoparticles, introduced into our daily lives as part of industrialization, urbanization, and modernization. These substances disrupt the epithelial barriers which leads to microbial dysbiosis and triggers a proinflammatory immune response to allergens, colonization by opportunistic pathogens, bacterial toxins, and autoantigens followed by chronic inflammation due to epigenetic mechanisms. Recent evidence from studies on the mechanisms of epithelial barrier damage has demonstrated that even trace amounts of toxic substances can damage epithelial barriers and induce tissue inflammation. Further research in this field is essential for our understanding of the causal substances and molecular mechanisms involved in the initiation of leaky epithelial barriers that cascade into chronic inflammatory diseases.

Household laundry detergents disrupt barrier integrity and induce inflammation in mouse and human skin

Rinaldi AO, Li M, Barletta E, D'Avino P, Yazici D, Pat Y, Ward S, Burla D, Tan G, Askary N, Larsson R, Bost J, Babayev H, Dhir R, Gaudenzio N, Akdis M, Nadeau K, Akdis CA, Mitamura Y. Allergy. 2024 Jan;79(1):128-141. doi: 10.1111/all.15891.

Background: Epithelial barrier impairment is associated with many skin and mucosal inflammatory disorders. Laundry detergents have been demonstrated to affect epithelial barrier function in vitro using air-liquid interface cultures of human epithelial cells.

Methods: Back skin of C57BL/6 mice was treated with two household laundry detergents at several dilutions. Barrier function was assessed by electric impedance spectroscopy (EIS) and transepidermal water loss (TEWL) measurements 4 hours after treatment with the detergent solutions. RNA sequencing (RNA-seq) and targeted multiplex proteomics analyses in skin biopsy samples were performed. The effect of the laundry detergent and sodium dodecyl sulfate (SDS) solutions after 6 hours was investigated on ex vivo human skin.

Results: Detergent-treated skin showed a significant EIS reduction and TEWL increase compared to untreated skin, with a relatively higher sensitivity and dose-response in EIS. The RNA-seq analysis showed a reduction in the expression of several genes essential for skin barrier integrity, such as tight junctions and adherens junction proteins. In contrast, keratinization, lipid metabolic processes, and epidermal cell differentiation were upregulated. Proteomics analysis showed that treatment with the detergent solutions generally downregulated cell adhesion-related proteins, such as epithelial cell adhesion molecule and contactin-1, and upregulated proinflammatory proteins, such as interleukin 6 and interleukin 1 beta. Both detergent and SDS significantly decreased EIS values in the ex vivo human skin model.

Conclusion: The present study demonstrated that laundry detergents and their main component SDS impaired the epidermal barrier in in vivo and ex vivo human skin models. Daily detergent exposure may impair the skin barrier function and may contribute to the development of atopic diseases.

B-cell immune response to human bocaviruses

Karaaslan C, Wirz O, Tan G, Globinska A, Boonpiyathad T, Hedman K, Vaselek S, Venermo MS, Jartti T, Akdis M, Akdis CA. Clin Exp Allergy. 2024 Jun;54(6):388-401. doi: 10.1111/cea.14453.

Background: Human bocaviruses (HBoVs) have been demonstrated to be implicated in respiratory and gastrointestinal infections, albeit there are limited studies on their immune response. In this study, we investigated the B cell immune responses to HBoV1 and HBoV2, representing two different species of bocaviruses in humans.

Methods: We analyzed the effects of stimulations with HBoV1 and 2 virus-like particles (VLPs) and of co-stimulation with HBoV1-rhinovirus (RV) on cells of the immune system by flow cytometry, transcriptomics, and luminometric immune assays.

Results: Human B cells, and particularly B regulatory cells (Breg cells), showed an increased immune response to HBoV1-VLPs stimulation. These immune responses were also supported by increased IL-1RA and PDL1 expressions in IL-10+ B cells from peripheral blood mononuclear cells (PBMCs) stimulated with HBoV1-VLPs. In addition, elevated levels of IL-10 and IL-1RA were found in the supernatants of peripheral blood mononuclear cells (PBMCs) following HBoV1-VLPs stimulation. HBoV1-VLPs and RV co-stimulation increased the IL-10+ B cell population. Transcriptome analysis by next-generation RNA sequencing showed an increased expression of IL-10 signalling and Breg cell markers in PBMCs stimulated with HBoV1-VLPs. Furthermore, TGF-β and chemo attractants MIP-1α, MIP-1β and IP10 protein levels were high in the supernatants of PBMCs stimulated with HBoV1-VLPs.

Conclusions: These findings demonstrate that in Breg cells, IL-10 signalling pathways and anti-inflammatory activity are induced by HBoV1, which can explain the often mild nature of the disease. In addition, the immune regulatory response induced by HBoV1-VLPs may indicate a potential immunomodulatory role of HBoV1 on the immune system and may represent a novel therapeutic target.

Davos May, 2025

Harvard Ehrenprofessur 2024
Prof. Dr. Cezmi Akdis, Prof. Dr. Mübeccel Akkdis and Prof. Dr. Kari Nadeau

Prof. Dr. Mübeccel Akdis

Immune Regulation
Role of Allergen-specific B cell in Allergy

Allergy is a globally spread affliction that is based on dysregulation of immune responses towards 'harmless' antigens. B cells and their regulation are also involved in mechanisms of allergic diseases. They produce the IgE essential for allergen-induced mast cell and basophil degranulation. The common consensus is that the regulation of B cells is closely tied to tolerance and allergy. In allergy, this regulation is likely dysfunctional, resulting in different B-cell behaviors. Therefore, we suspected that B cells from allergic individuals may differ in their class-switching potential or process, making them more prone to IgE class-switching and allergies. We hypothesized that there would be significantly different expressions in pathways related to B-cell activation, B-cell regulation or B-cell isotype switching correlating to allergic symptoms. We had 16 twin pairs that were either healthy, allergy discordant or allergy concordant. The donors were monozygotic and mainly allergic to timothy grass, birch tree pollen and/or house dust mites; none had taken or were on allergen-immunotherapy. We sorted switched and unswitched B cells from bio-banked PBMCs, extracted RNA, and performed 100 bp single-end RNA sequencing on the Illumina Novaseq 6000 platform.

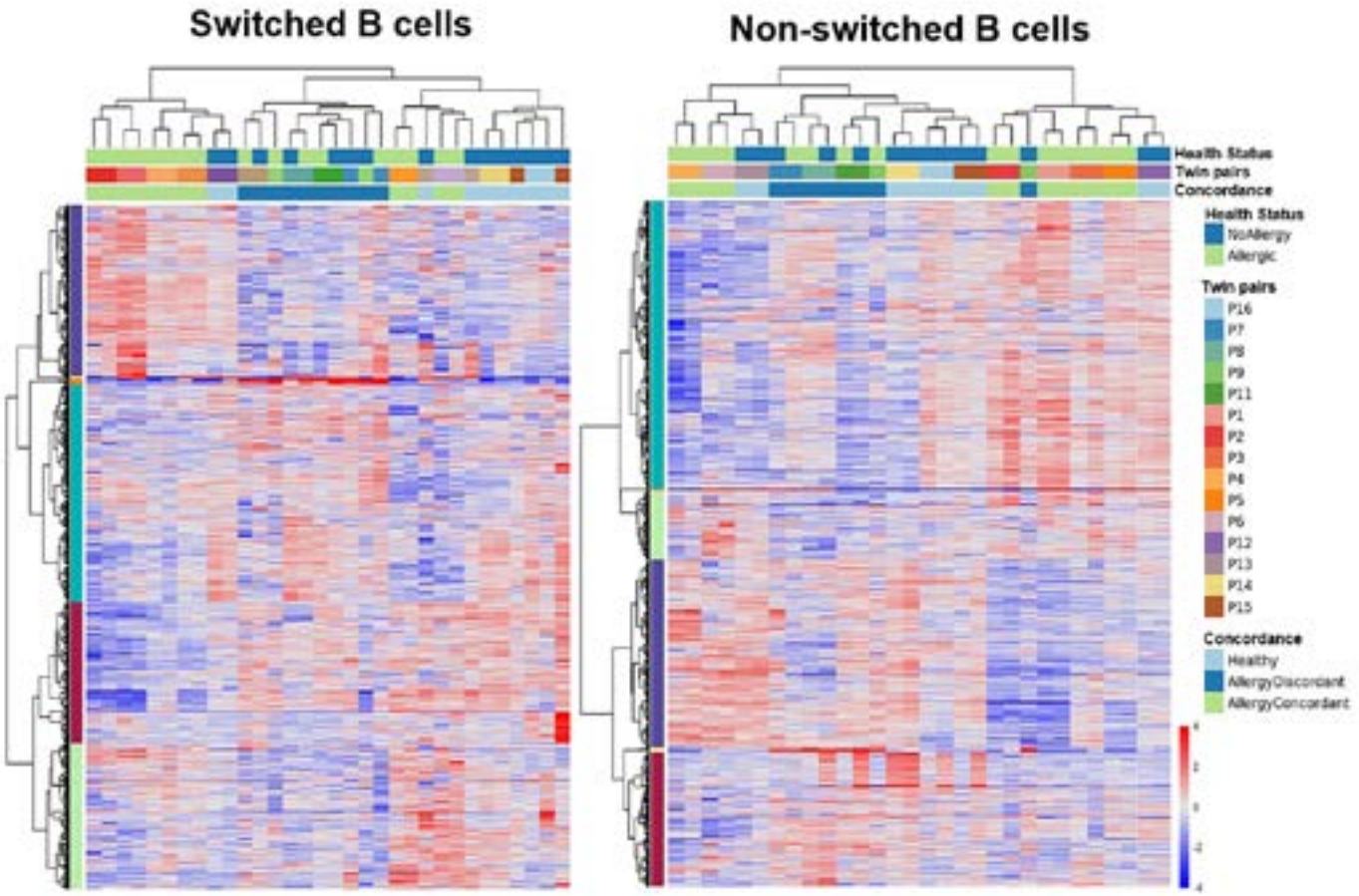


Figure 1. (A) Heatmap of the bulk RNA sequencing for non-switched and switched B cells. The three bars on top of the graph depict the three conditions relevant for cluster analysis. The first Bar depicts the health status of each individual donor while the second bar labels the donors of each twin pair by the same color. The third bar depicts in which concordance group the twins belongs to. Significant genes have an absolute fold change >0.5 and a p-value <.05. The bars show clustering by concordance in switched and clustering by twin pair in unswitched B cells.

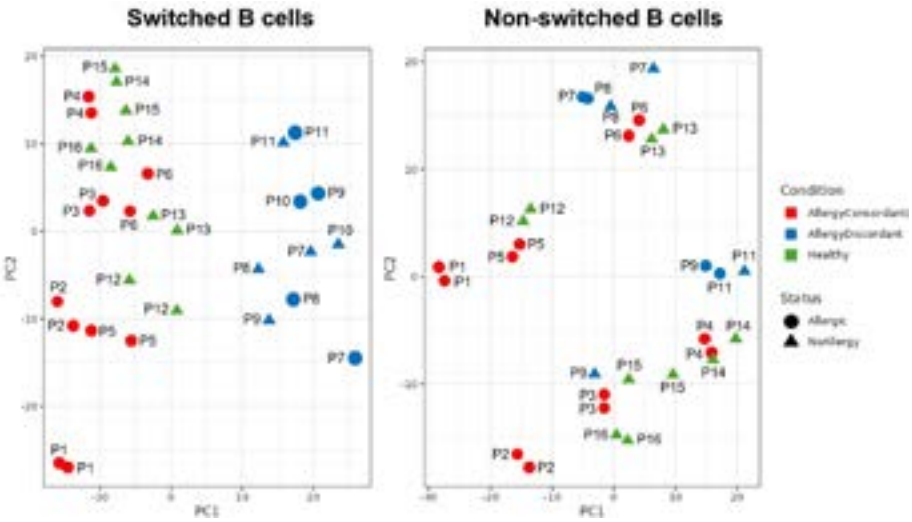


Figure 1. (B) PCA of the top 300 significant genes (p-value) for all samples. Circles depict allergic and triangles healthy individuals. The colors identify the concordance group of each donor. The labels describe which twins are a pair. The PCA shows the how strong the batch effects in switched B cells and twin pairings in the unswitched B cells influence the clustering of the data set.

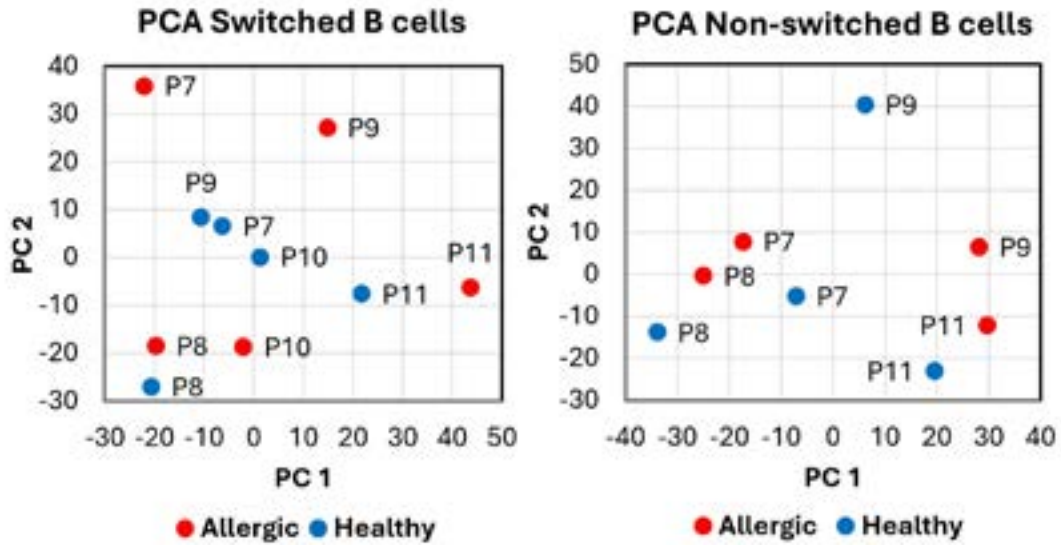


Figure 2. (A) PCA of the top 300 significant genes in allergy discordant twins. Data labels indicate which twins are a pair. The analysis shows that healthy and allergic siblings do not cluster in groups but are spread out homogenously. Non-switched B cells split in two groups along the PC 1 axis, which is also reflected in the STRINGR web pathway analysis.

Unbiased clustering of samples (Figure 1A) showed that the main influences for clustering in descending order are switched versus non-switched B cells, twin pairs and then their concordance status. The degree of clustering by concordance status varies, yet there is no clustering by allergy within the discordance clusters in both switched and non-switched B cells, suggesting that it is a confounding factor. An individuals' allergy status had little to no significant impact on the overall clustering. The same holds true for the PCA analysis of the top 300 significant genes of switched B cells, while non-switched B cells are separated by gender (Figure 1B). Comparing healthy concordant to allergy concordant twins could be greatly influenced by confounding factors like twin pair similarities and grouping by concordance instead of their actual health status. Therefore, we compared the healthy versus allergic within the discordant twin pairs to avoid these influences for gene expression analysis. In this comparison, neither switched nor non-switched B cells show pathways that would traditionally be associated with allergies or B-cell regulation. We did not find significant differences in pathway regulations on the wider scale of B cells between allergy-discordant twins. PCA analysis for the top 300 genes by the p-value of the allergy-discordant twins confirmed that allergic versus healthy twins do not group by allergy status (Figure 2A). Pathway analysis of the top genes in allergy-discordant twins does not reveal any cohesive pathways (allergy vs. healthy) in the switched B cells (Figure 2B). One pathway in the non-switched B cells upregulated genes, is associated with the cell cycle but not directly with immune functions. Our results show no indication that there is a general dysregulation of B cells as an underlying cause for allergies. Any differences that might exist are too subtle to be observed across B cells. We propose that distinctions between allergic and non-allergic individuals may only be noticeable in allergen-specific B cells. These effects are probably overshadowed by the variability among individuals due to the rarity of allergen-specific B cells in the overall B-cell population. Since the study focused on PBMCs, tissue-resident B cells may exhibit different properties.

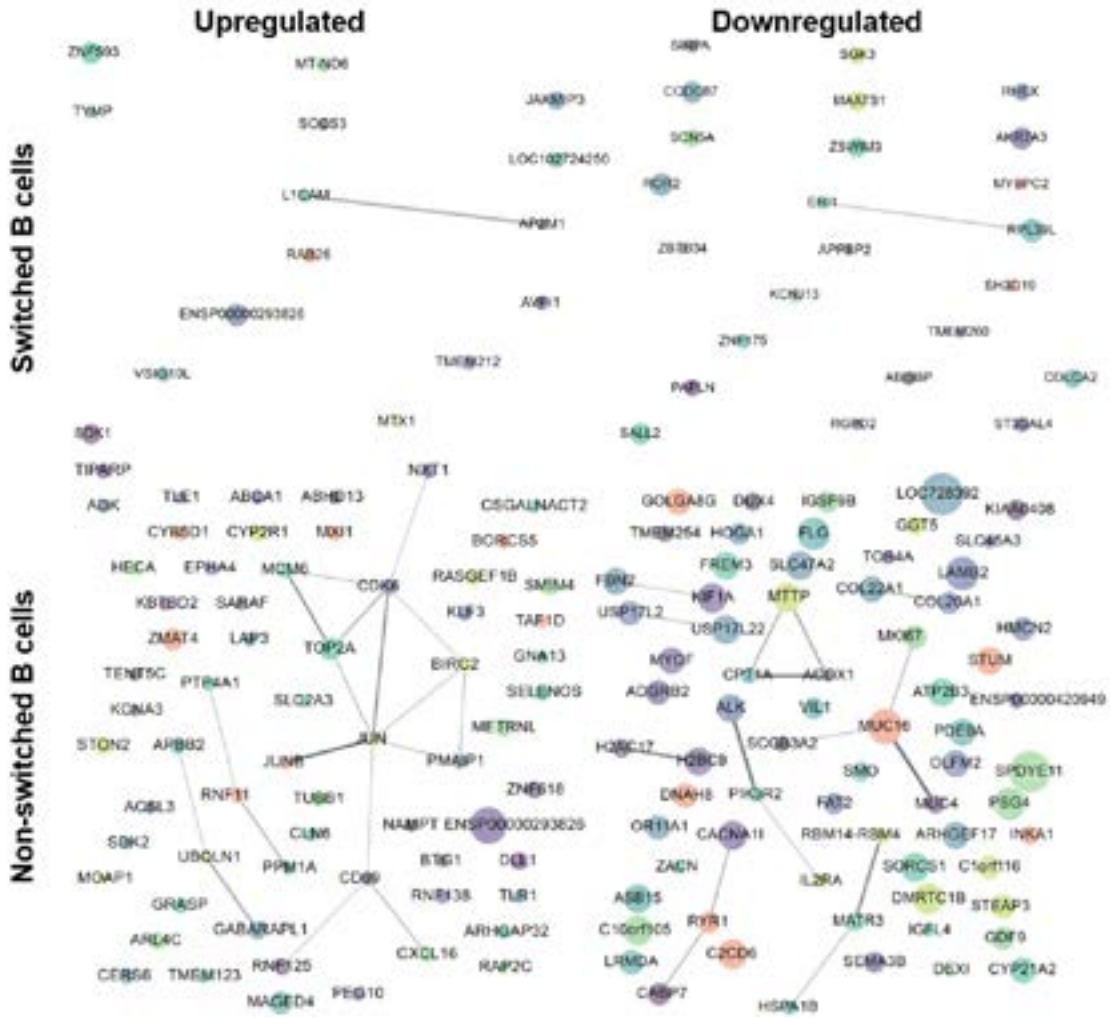


Figure 2. (B) Pathway analysis of the top 300 up and downregulated genes by p-value. The circle size of each gene reflects the log2 fold change in expression. Line strength indicates edge confidence according to Stringr pathway analysis. The pathway analysis shows allergic samples compared to healthy samples.

Immunological and Allergen-Specific B cell Tolerance

Immunological tolerance refers to an active immune system response to specific antigens. Allergen tolerance development is often associated with an increase in allergen-specific regulatory T (Treg) and B (Breg) cells, heightened production of allergen-specific IgG4 antibodies, and reduced activation of effector cells such as basophils, mast cells, and eosinophils. B cells play a crucial role in the pathogenesis of allergies by producing antigen-specific IgE and contribute to allergen tolerance. They do so primarily through immunosuppressive cytokines like IL-10, TGF-β, and IL-35, which induce Breg cells and promote the production of protective IgG4 isotypes. Extensive research on allergen tolerance has been conducted using models of natural exposure to high allergen doses, such as in beekeepers, cat owners, and individuals with helminth infections. Beekeepers, for instance, experience frequent stings during the active season but have limited exposure during autumn and winter. Studies on bee venom allergen-specific T and B cells in healthy beekeepers have significantly advanced understanding of the mechanisms that induce allergen tolerance. High-dose exposure in beekeepers has been linked to reduced T-cell-mediated cutaneous late-phase swelling, increased IL-10-producing Tr1 cells, and elevated allergen-specific IgG4 levels.

Food Allergy and Oral Immunotherapy

Food allergy (FA) is a significant public health concern, affecting up to 10% of the global population. Cow's milk allergy (CMA) is the most common FA in young children, with about 50% achieving "natural tolerance" by age five. However, persistent CMA is often associated with multiple allergic comorbidities and a more severe clinical presentation. Oral immunotherapy (OIT) has emerged as a treatment strategy to induce desensitization and remission to the sensitizing allergen. OIT involves gradually introducing small amounts of allergenic food (e.g., milk, peanut, or egg) into the diet, leading to desensitization and, ideally, longer-term remission (formerly termed "sustained unresponsiveness"), enabling the individual to consume the food freely.

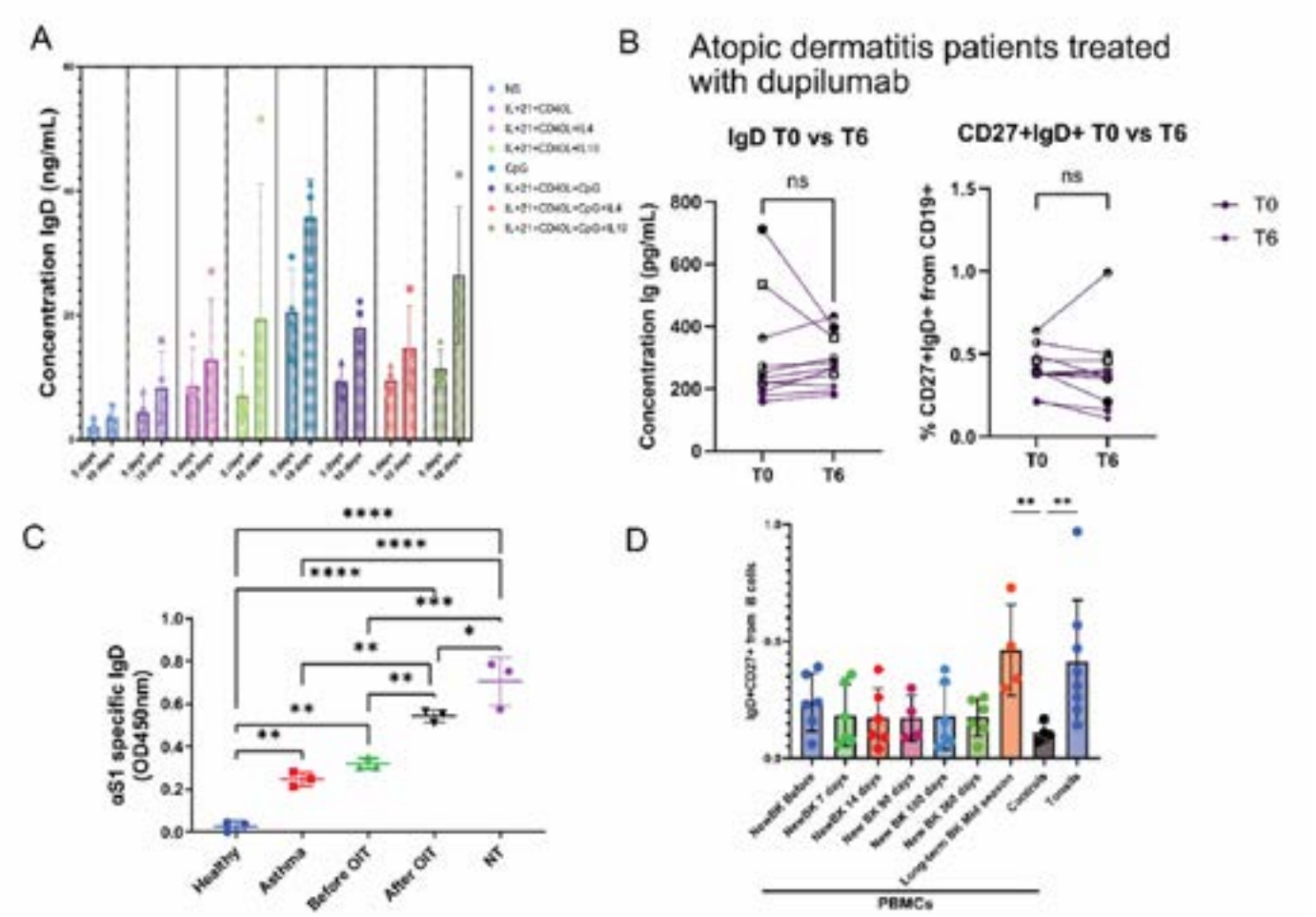


Figure 3. The role of IgD in immune tolerance. (A)Purified B cells in presence of CpG and IL4 together with IL21+sCD40L increase the production of total of IgD. (B) Treatment with anti-IL4Rα did not affect the production of IgD neither memory IgD expressing cells.(C) Individuals with natural tolerance and after OIT increase the levels of IgD against cow's milk allergen αs1-casein. (D) Long-term exposed beekeepers and in tonsillar mononuclear cells, memory IgD is higher compared with new exposed beekeepers and controls.

Understanding the immunological factors driving OIT responses and identifying biomarkers to distinguish responders, non-responders, and remission remains a critical challenge. Our recent study, titled "Allergen-specific B cell responses in oral immunotherapy-induced desensitisation, remission, and natural outgrowth in cow's milk allergy", explored the trajectory of allergen-specific B cells during OIT. The study uniquely compared findings in individuals achieving desensitization or remission with those naturally tolerating the allergen. The research revealed that Bos 9-specific B cells were present across all patient groups, challenging the assumption that allergen-specific cells are confined to allergic individuals. In healthy controls, Bos 9-specific B cells were proficient in producing sIgG1 and sIgG4 but lacked sIgE production capability. Transcriptomic analysis highlighted distinct gene signatures between desensitized and remission groups, establishing different changes according to the stage. Key findings included exclusive upregulation of IL10RA and TGFB3 in desensitized OIT patients, while genes related to B cell activation (RIF1, FOXP1, JAK3), BCR signaling (PIK3CA, PIK-3CD, PRKCB), and differentiation (BCL11A, CARD11, DOCK11, MALT1) were downregulated in both desensitized and naturally tolerant individuals. Genes such as TLR4, IGHG1, and IGHA2 were significantly upregulated in remission patients, yet changes did not entirely overlap with natural tolerance profiles. Natural tolerance was characterized by increased cell activation (STAT6, FOXP1, NOTCH2, TLR9), cytokine-related genes (IL2RG, IL7, AHR), B cell differentiation (BCL6, CARD11, DOCK11), and BCR signaling (CD22, PIK3CA).

Insights into IgD and Immune Tolerance

Immunoglobulin D (IgD) has recently emerged as a potential key regulator in allergy and tolerance responses. Our ongoing research investigates the role of IgD in allergy, natural tolerance, and high-dose allergen exposure models. In studies involving cow's milk OIT cohorts, atopic dermatitis patients treated with dupilumab (anti-IL4Rα), and beekeepers exposed to bee venom, significant findings have been observed. The CD19+IgD+IL-10+ population increased in PBMCs after CpG stimulation, and IgD+IgM- B cells co-expressed CD22 (a BCR signaling inhibitor) and Notch2 (associated with marginal zone B cell development). However, IL-10 expression was limited to IgM-expressing cells.
Stimulation with CpG or IL-21+sCD40L+IL-4 enhanced IgD produc-

tion in purified B cells, though dupilumab treatment did not affect IgD levels or CD27+IgD+ B cells after six months. In the cow's milk cohort, αS1-casein-specific IgD increased significantly in OIT and natural tolerance groups compared to pre-OIT levels. Notably, CD27+IgD+ B cells were more prevalent in long-term allergen-exposed beekeepers and tonsillar mononuclear cells (TMCs) compared to new beekeepers and controls (Figure 3). These findings highlight IgD's potential role in establishing immune tolerance, though further studies are needed to clarify specific mechanisms.

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. 2024 Jul 11. doi: 10.1111/all.16220. Online ahead of print. 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.

Active human ascariasis increases frequencies of circulating activated type-2 peripheral blood innate lymphoid cells and tryptase AB1 levels.

(Juan-Felipe Lopez, Josefina Zakzuk, Pattraporn Satitsuksanoa, Ana Lozano, Laura Buergi, Anja Heide, Juan Alvarado2, Huseyn Babayev, Cezmi Akdis, Willem van de Veen, Luis Caraballo, Mübeccel Akdis, Front Immunol. 2024 Oct 25;15:1459961.)

Ascaris lumbricoides infection is one of the most common soil-transmitted helminthiases. IgE response to this helminth may increase the risk of asthma, bronchial hyperreactivity, and atopy. There is evidence showing the role of type-2 innate lymphoid cells (ILC2) in the pathogenesis of helminth infection and allergy. However, research on human Ascaris infection is scarce. The aim of the study to investigate and characterize the influence of helminth infection by Ascaris lumbricoides on circulating ILCs from subjects endemically exposed. Non-infected (NI; n=16) and Ascaris-infected (AI; n=16) subjects from an endemic area were included. Two consecutive stool samples from each subject were examined by Kato-Katz to define parasite infection. Antibodies to ABA-1 (an antigen from Ascaris) and Ascaris extract were measured by ELISA. Frozen PBMCs were received and ILCs, together with activation markers (CD25, CD69, thymic stromal lymphopoietin receptor (TSLPR) and NKp44), were evaluated by flow cytometry. Proximity extension assay (PEA) was performed to explore proteins associated with the infection. No significant differences in the relative frequency of total ILCs, ILC1, ILC2 and ILC3 were observed regarding to infection status. However, within AI group, ABA-1-IgE-sensitized subjects had higher frequencies of ILC2 ($p<0.05$). Frequencies of activated ILC2 (CD25+, CD69+, and TSLPR+) were higher in AI compared to the NI ($p<0.01$). Additionally, egg burden was positively correlated with CD69+ ILC2 frequencies ($r=0.67$; $p=0.005$). Tryptase alpha/beta 1 (TPSAB1), and several proteins associated with granulocyte chemotaxis were highly expressed in the AI group ($p<0.05$). Interestingly, TPSAB1 levels were positively correlated with activated-ILC2 frequencies and IgE levels against Ascaris extract. Ascaris infection is associated with increased activation of ILC2 and TPSAB1 and these factors could contribute to its type-2 response boosting effect.

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. 2024 Oct 29. doi: 10.1111/all.16376. Online ahead of print. PMID: 39470621 No abstract available.)

Davos, May 2025

Precision Proteomics Center

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 as a Leading House, recognizing proteomics as a key technology in life sciences. The center aims to develop a nationally and internationally recognized research platform for cutting-edge proteome research. Fully integrated into SIAF's infrastructure, the Precision Proteomics Center fosters collaboration and scientific excellence within SIAF and its partner institutes.

Since 2022, Christoph Messner has led the Proteomics Center Davos while holding a professorship at the University of Zurich. The center is dedicated to integrating proteomics with precision medicine, driving technological innovation, and advancing translational research. Strategically aligned with SIAF, other research institutes in the canton, and the University of Zurich, it plays a key role in strengthening proteomics research in Switzerland.

At the Precision Proteomics Center, we develop mass spectrometry-based proteomics workflows with a specific focus on enhancing their robustness and efficiency for clinical applications. In addition to measuring protein abundances, we focus on developing workflows that utilize peptide-level information and analyze post-translational modifications, particularly glycosylations. We apply these technologies in biomarker discovery studies of large cohorts, as well as in functional studies in various disease areas, with a particular focus on allergies, skin diseases, and oncology.

Proteins are the functional building blocks of life, and their levels are defined by both genetic and environmental factors. The precise regulation of proteins is fundamental in all biological processes and alterations of the proteome are associated with diseases. Thus, studying proteins and its association with the phenotype is key for cell biology, understanding disease mechanisms, and ultimately improving patient care.

Skin Proteomics – Unraveling Disease Mechanisms and Advancing Diagnostic Applications

Inflammatory skin diseases such as atopic dermatitis are influenced by complex interactions between genetic and environmental factors. Over the past year, we have advanced our skin proteomics platform to address persistent challenges in molecular profiling of skin samples.We successfully applied non-invasive sampling methods, particularly tape stripping, across several cohortsThis approach has proven effective in capturing disease-relevant molecular signatures without the need for invasive biopsies.Our analyses have revealed promising protein markers associated with disease activity and genotype, moving us closer to developing objective, quantifiable tools for diagnosis and therapy monitoring.

Precision Proteomics in Lymphoma Research

Lymphomas comprise a highly heterogeneous group of cancers, with over 80 distinct subtypes, each exhibiting unique clinical behaviors and treatment responses. These complexities pose significant challenges in diagnosis and therapy. Current diagnostic approaches primarily rely on morphology, immunophenotyping, and genetics. However, with a growing arsenal of therapeutic options, a deeper understanding of disease mechanisms and more personalized treatment guidance are urgently needed.
In collaboration with the University Hospital Zurich Biobank (led by Thorsten Zenz and Marco Bühler), we have made significant progress within our LOOP Zurich-funded project aimed at enhancing lymphoma diagnostics through proteomics.Over the past year, we have begun processing and analyzing a cohort of more than 2,500 tissue and liquid biopsy samples, representing a broad spectrum of lymphoma subtypes. This effort will result in the most comprehensive proteomic dataset to date in the field of lymphoma research. These advances are bringing us closer to implementing proteomics-based diagnostics and precision oncology at the University Hospital Zurich.

Glycosylation in Allergies and Immune Regulation

At the Proteomics Center in Davos, we have expanded our technological capabilities to explore the role of glycosylation in immune regulation and allergic diseases. Glycosylation remains an underexplored yet critical layer of molecular information.

This year, we developed and refined mass spectrometry-based workflows tailored for glycan analysis. Our focus has been on IgE antibodies, which are central to allergic responses, and how their glycosylation patterns relate to disease severity and treatment efficacy.

These efforts are beginning to uncover novel glyco-biomarkers and molecular mechanisms that could enhance allergy diagnostics and open new therapeutic avenues.

SIAF Annual Report 2024 28

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 (accepted)

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.

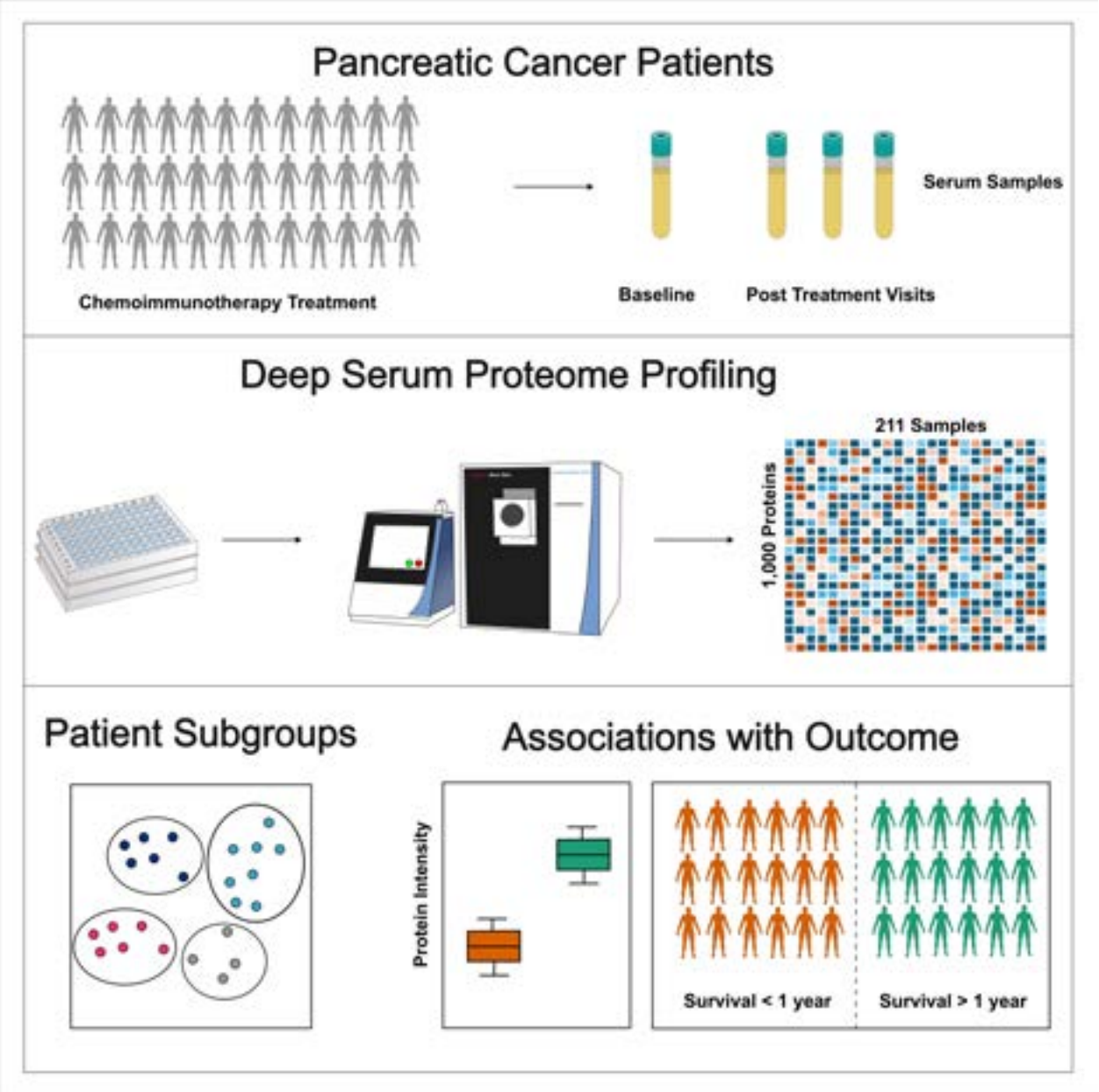


Figure 1: Serum proteomics identifies patient subgroups and predicts survival in PDAC patients receiving immunotherapy combined with chemotherapy.

Precision Proteomics Center

Oxonium ion scanning mass spectrometry for large-scale plasma glycoproteomics.
White ME, Sinn LR, Jones DM, de Folter J, Aulakh SK, Wang Z, Flynn HR, Krüger L, Tober-Lau P, Demichev V, Kurth F, Mülleder M, Blanchard V, Messner CB, Ralser M*. Nature Biomedical Engineering. 2024 Mar;8(3):233-47, *These Authors contributed equally.*
Research Briefing: Messner CB, Ralser M. Nature Biomedical Engineering. 2024 Mar 1;8(3):212-3.

Protein glycosylation, a complex and heterogeneous post-translational modification that is frequently dysregulated in disease, has been difficult to analyse at scale.

Here we report a data-independent acquisition technique for the large-scale mass-spectrometric quantification of glycopeptides in plasma samples. The technique, which we named 'OxoScan-MS', identifies oxonium ions as glycopeptide fragments and exploits a sliding-quadrupole dimension to generate comprehensive and untargeted oxonium ion maps of precursor masses assigned to fragment ions from non-enriched plasma samples. By applying OxoScan-MS to quantify 1,002 glycopeptide features in the plasma glycoproteomes from patients with COVID-19 and healthy controls, we found that severe COVID-19 induces differential glycosylation in IgA, haptoglobin, transferrin and other disease-relevant plasma glycoproteins. OxoScan-MS may allow for the quantitative mapping of glycoproteomes at the scale of hundreds to thousands of samples.

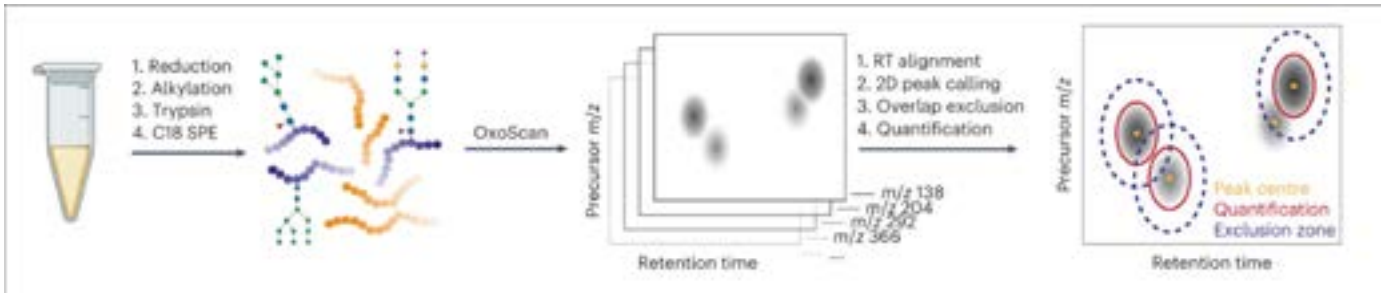
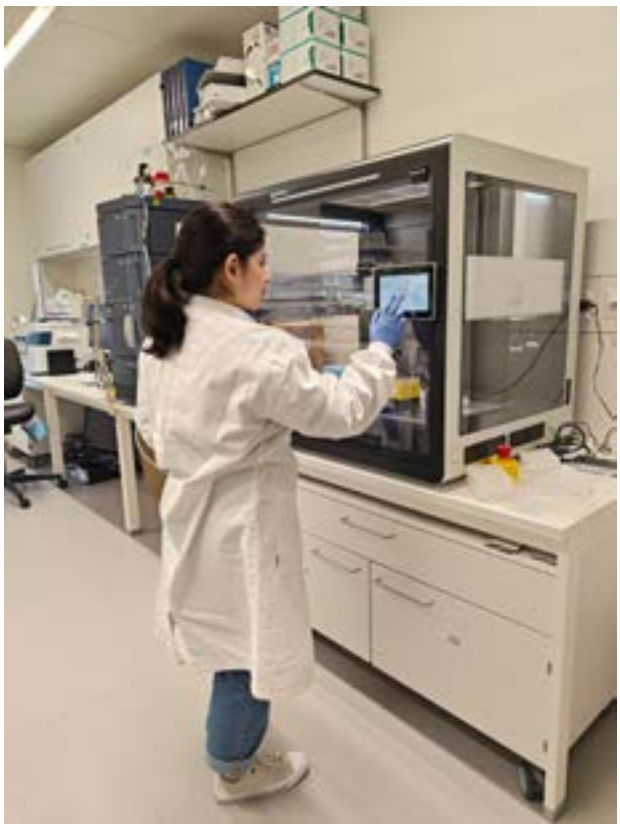


Figure 2: Oxonium ion scanning enables the quantification of glycopeptides across large-scale cohorts..

Persistent complement dysregulation with signs of thromboinflammation in active Long Covid
Cervia-Hasler C, Brüningk SC, Hoch T, Fan B, Muzio G, Thompson RC, Ceglarek L, Meledin R, Westermann P, Emmenegger M, Taeschler P., Zurbuchen Y, Pons M, Menges D, Ballouz T, Cervia-Hasler S, Adamo S, Merad M, Charney AW, Puhan M, Brodin P, Nilsson J, Aguzzi A, Raeber M, Messner CB, Beckmann ND, Borgwardt K, Boyman O Science. 2024 Jan 19;383(6680):eadg7942.

Long Covid is a debilitating condition of unknown etiology. We performed multimodal proteomics analyses of blood serum from COVID-19 patients followed up to 12 months after confirmed severe acute respiratory syndrome coronavirus 2 infection. Analysis of >6500 proteins in 268 longitudinal samples revealed dysregulated activation of the complement system, an innate immune protection and homeostasis mechanism, in individuals experiencing Long Covid. Thus, active Long Covid was characterized by terminal complement system dysregulation and ongoing activation of the alternative and classical complement pathways, the latter associated with increased antibody titers against several herpesviruses possibly stimulating this pathway. Moreover, markers of hemolysis, tissue injury, platelet activation, and monocyte–platelet aggregates were increased in Long Covid. Machine learning confirmed complement and thromboinflammatory proteins as top biomarkers, warranting diagnostic and therapeutic interrogation of these systems.

Davos, May 2025



SIAF Annual Report 2024 30

PD Dr. Katja Baerenfaller

Challenging the current view on protein prenylation in T helper cells

Concluding Jana Koch's PhD project that was funded by "Stiftung vormals Bündner Heilstätte Arosa" and by the Center for Precision Proteomics, we have published the review article "Differenzierung und Aktivierung von Th1-Zellen – ein Multi-omics Ansatz" in Dermatologie Praxis, and is part of the CME programme for medical doctors.

Graduation ceremony of Damir Zhakparov at the University of Zurich on 13.12.24 with Dr. Katja Bärenfaller and Prof. Dr. Roland Sigel, Dean of the Science Faculty

In this article, we explored the central role of Th1 cells, which are key components of the adaptive immune response and are involved in a wide range of diseases (Figure 1). We also highlighted that the regulation of cellular processes occurs at all levels of protein biosynthesis, underscoring the complexity of immune cell function. To address this complexity, the integration of multiple omics technologies, such as epigenomics, transcriptomics, translatomics, and

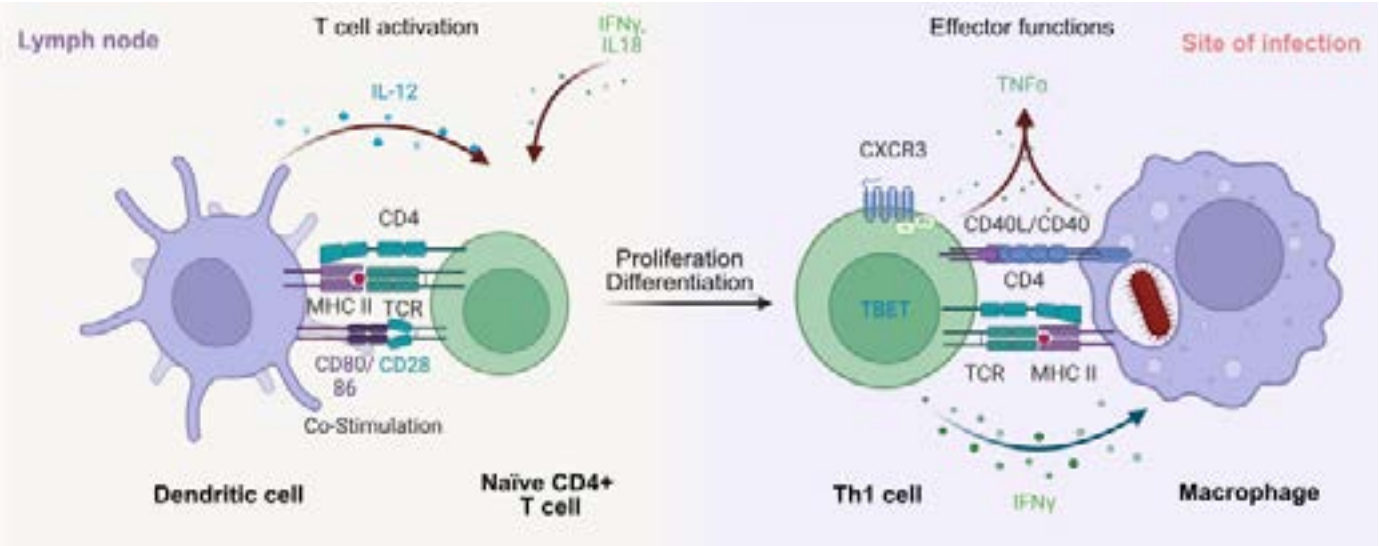


Figure 1: *Adapted from Figure 1 in Koch and Baerenfaller, Differenzierung und Aktivierung von T-Helfer 1-Zellen, ein Multi-omics-Ansatz, Dermatologie Praxis 2024; Vol. 23, Nr.2, DOI: 10.5167/uzh-275686*

proteomics, enables more detailed and comprehensive analyses of Th cells. (Koch and Baerenfaller, 2024).

As part of this project, we also summarised our experimental data and bioinformatics analyses on protein prenylation in Th1 cells. Protein prenylation is a posttranslational modification in which a prenyl group, either a farnesyl (C15) or geranylgeranyl (C20) moiety, is attached to cysteine residues in proteins. This lipid modification increases the hydrophobicity of proteins and enhances their affinity for membranes, thereby influencing their subcellular localisation and molecular interactions.

According to current consensus, prenylation primarily occurs at defined motifs located at the protein C-terminus, primarily at CaaX or CXXX motifs. However, our data challenge this view by identifying both additional C-terminal prenylation motifs and internal prenylation sites (Figure 2AB). Notably, these internal sites appear to follow structural rather than strictly sequence-based motifs. These findings suggest that current annotation criteria for prenylated proteins should be revised to include non-canonical and structurally defined sites

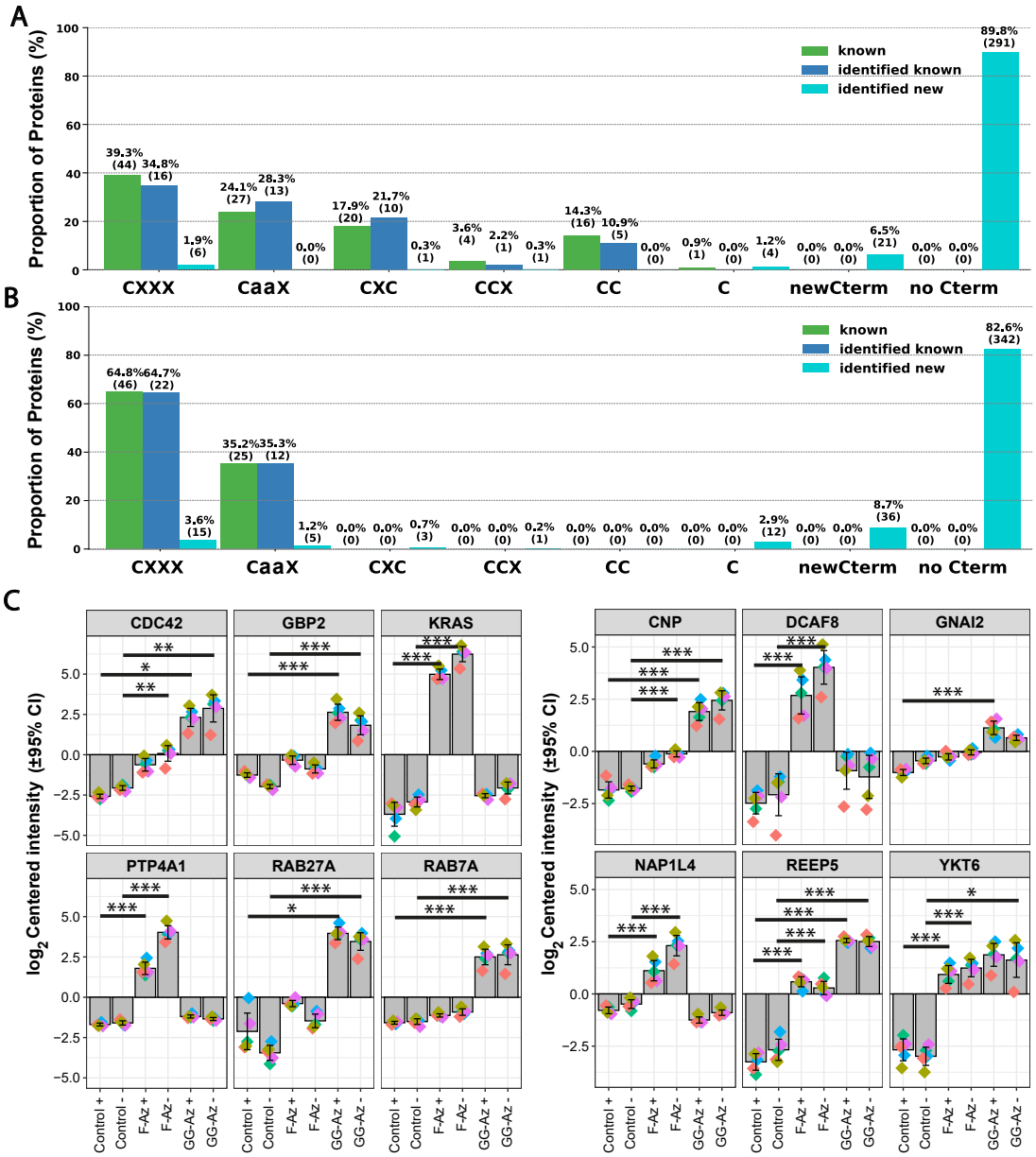


Figure 2: *Figure 2 as published in the preprint (Koch et al., DOI: 10.21203/rs.3.rs-6305485/v1). Proportion of known prenylated proteins (green), known prenylated proteins identified in our study (blue), and proteins identified here that were not previously known to be prenylated (turquoise), classified according to the prenylation motif found in their sequence. The classification includes known motifs, newly identified C-terminal motifs, and proteins lacking a C-terminal cysteine for A) samples treated with farnesyl alcohol azide (F-Az) and B) samples treated with geranylgeranyl alcohol azide (GG-Az). C) Log2-centred intensity values of proteins identified as prenylated in activated (+) and non-activated (-) Th1 cells treated with F-Az, GG-Az, or mock-treated in the controls. N= 5 donors per condition. Diamonds represent individual donors * = p<0.05, ** = p< 0. 1, *** = p< 0.005.*

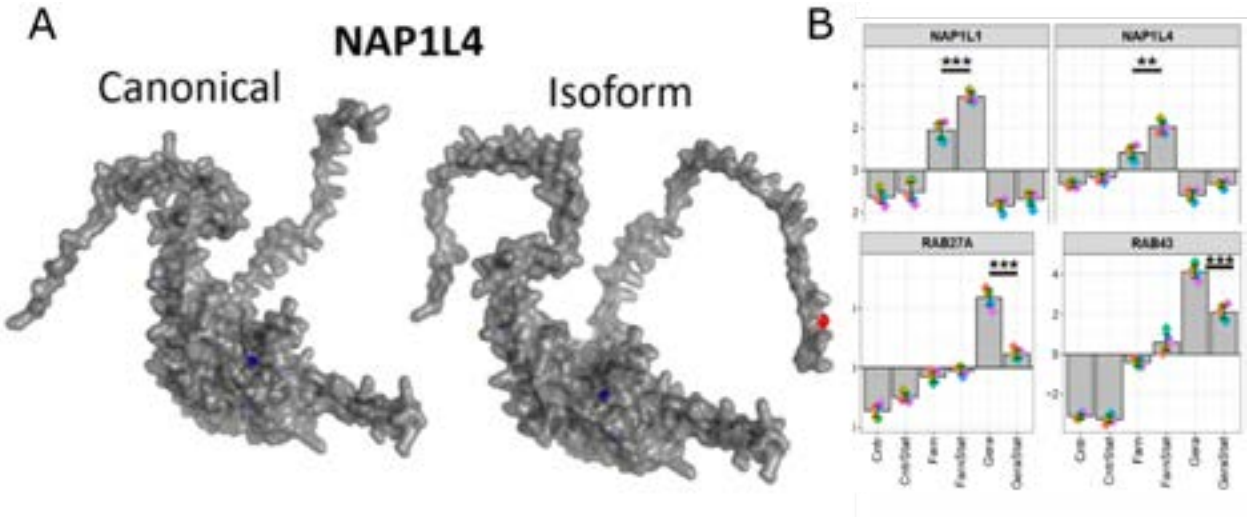


***Figure 3**: 3D structures of NAP1L4, displaying the canonical form as predicted by AlphaFold2 (left) and an isoform with a C-terminal cysteine modelled by SWISS-MODEL (right). B) Log2-centred intensity values of proteins identified as prenylated in activated Th1 cells pretreated or not with statin and treated with F-Az, GG-Az, or mock-treated in the controls (Cntr). N= 5 donors per condition. Diamonds represent individual donors * = p<0.05, ** = p< 0. 1, *** = p< 0.005.*

In our analyses, we identified several proteins that are already known to be prenylated, as well as novel prenylated proteins (Figure 2C). For some of these proteins, including REEP5 and NAP1L4, the canonical isoforms did not contain a cysteine within a recognised prenylation motif, whereas alternative isoforms did. Interestingly, the canonical isoform of NAP1L4 lacks a large C-terminal arm that is present in the alternative isoform and contains a C-terminal CXXX prenylation motif (Figure 2A). This arm is also present in the known farnesylated protein NAP1L1.

Further supporting the notion that both NAP1L1 and NAP1L4 are farnesylated is the finding that pretreatment with statin, followed by supplementation of the cells with F-Az, led to increased farnesylation. This statin-induced enhancement was also observed for other farnesylated proteins following F-Az supplementation, which was expected, as statin inhibits the mevalonate pathway and thereby deprives cells of the prenyl moieties synthesised via this pathway. Interestingly, the opposite effect was observed for geranylgeranylated proteins of the RAB family, which are thought to be modified by Rab geranylgeranyltransferase. These results may therefore be explained by off-target effects of pitavastatin, potentially involving inhibition of Rab geranylgeranyltransferase. Given the widespread use of statins to lower cholesterol levels, such off-target effects could be biomedically relevant and certainly warrant further investigation.

Identification of Relevant Features in Complex Biomedical Datasets Using Artificial Intelligence

This was the title of the cumulative PhD thesis of Damir Zhakparov, the PhD student involved in several projects at the Cantonal Center for Data Analysis, Visualisation and Simulation (DAViS). Damir Zhakparov successfully defended his thesis on 21 August 2024.

In the concluding phase of his work, and building on previous efforts, we focused intensively on integrating variables from four datasets: selected features from RNA sequencing data, clinical data, cytokine profiles, and antibody measurements. Using the DIABLO model, we examined how variables across these datasets interacted and identified three distinct clusters of correlated features from different datasets. Notably, two of these clusters were associated with AD susceptibility clusters, while one emerged as a protective cluster, comprising cytokines and clinical questionnaire features such as exposure to sunlight and contact with farm animals (Zhakparov et al., manuscript in preparation).

Unlocking Molecular Insights into Sports Medicine using Proteomics

Proteomics is transforming sports medicine by addressing key questions such as recovery from training and molecular differences between athletes in different disciplines. A crucial aspect of serum proteomics is the analysis of extracellular vesicles, small bodies in body fluids that carry proteins, nucleotides, and other biomolecules. These vesicles play a vital role in cell communication and may reveal novel biomarkers for performance and recovery.

In collaboration with the Swiss Research Institute for Sports Medicine (SRISM), we investigate various aspects of sports medicine using a combination of targeted and non-targeted mass spectrometry-based proteomics across different cohorts of elite athletes. For example, we analyse total serum and serum extracellular vesicles from female elite ice hockey players to gain molecular insights into training and recovery (Barletta, Rüegg et al., manuscript in preparation).

Davos, May 2025

Vaccine Development

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. We aimed to identify and characterise Aspergillus antigen-reactive T cells in EA and to analyze immunoglobulin (Ig) isotypes in EA, elucidating their binding to different A. fumigatus antigens, and their quantities systemically in serum and locally in bronchoalveolar lavage fluid (BALF).
MEA and SEA differed from HE but hardly from each other. Compared to HE, asthmatic horses showed increased anti-A. fumigatus binding of IgG (BALF and serum) and IgA (BALF). Serum and BALF IgE binding and total IgE contents were similar between HE and EA. Single antigens, as well as A. fumigatus lysate, yielded similar Ig binding patterns. Serum and BALF IgG1 binding to all antigens was increased in SEA and to several antigens in MEA. Serum IgG4/7 binding to two antigens was increased in SEA. BALF IgA binding to all antigens was increased in SEA and MEA. Total BALF IgG1 and IgG4/7 contents were increased in SEA, and serum IgG4/7 content was increased in MEA compared to HE. Yet, total isotype contents differentiated EA and HE less clearly than antigen-binding Ig.
Jentsch et.al. Front Immunol. . 2024 Jun 17:15:1406794 doi: 10.3389/fimmu.2024.1406794. eCollection 2024.
Wjst et. al. Front Immunol. . 2024 Aug 20:15:1367971. doi: 10.3389/fimmu.2024.1367971. eCollection 2024.

Topic: Atopic dermatitis

Skin barrier dysfunction is associated with the development of atopic dermatitis (AD), however methods to assess skin barrier function are limited. We investigated the use of electrical impedance spectroscopy (EIS) to detect skin barrier dysfunction in children with AD of the CARE (Childhood AlleRgy, nutrition, and Environment) cohort.
Based on the EIS/AD score, the EIS algorithm was able to clearly discriminate between healthy skin and clinically unaffected skin of children with active AD (area under the curve 0.92, 95% CI 0.85-0.99). It was also able to detect a difference between healthy skin and AD skin when the child did not have active AD. There was no clear association between the EIS/AD score and the severity of AD or sensitization to the tested allergens. The performance of the algorithm was not affected by age
Sasaki et. al. Allergy. 2024 Jan; 79(1):142-152. doi: 10.1111/all.15895. Epub 2023 Sep 27.

Davos, May 2025

Graubünden forscht 2024

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, 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 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 (Figure 1), 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 gene editing, multi-color flow cytometry, 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, 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.

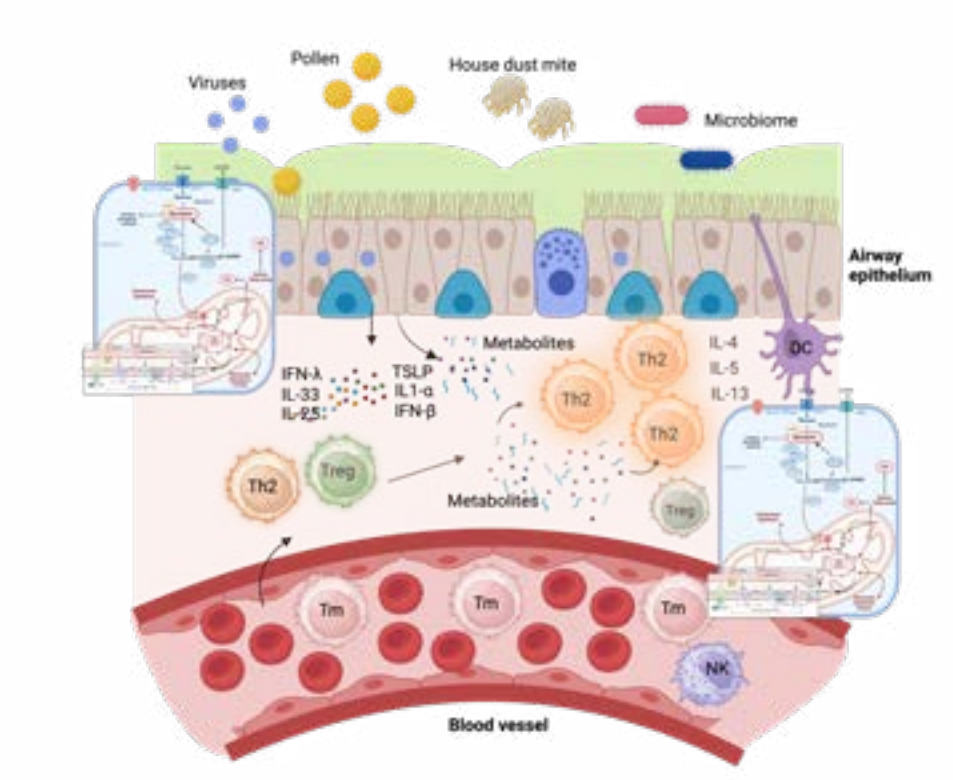


Figure 1. Research focus: We study how microenvironment in the lung in allergic asthma, induced by repeated allergen exposure, viral infection or microbiome dysbiosis, might affect metabolism, mitochondria and immune functions of airway epithelium and infiltrating Th2 and Treg cells, and how it may further lead to inefficient antiviral response and increased type 2 inflammation. We also study other mucosal interfaces in the gut, joints and skin.

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

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. Epub 2024 Oct 28.

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.

A survey study on antibiotic prescription practices for acute asthma exacerbations: An European academy of allergy and clinical immunology task force report

Redel AL, Feleszko W, Arcolaci A, Cefaloni F, Atanaskovic-Markovic M, Braunstahl GJ, Boccabella C, Bonini M, Karavelia A, Louwers E, Mülleneisen N, O'Mahony L, Pini L, Rapiejko A, Shehu E, Sokolowska M, Untersmayr E, Tramper-Stranders G; EAACI Task Force on Conscious and Rational use of Antibiotics in Allergic Diseases. Clin Transl Allergy. 2024 Mar;14(3):e12345. doi: 10.1002/clt2.12345.

Guidelines recommend treating asthma exacerbations (AAEs) with bronchodilators combined with inhaled and/or systemic corticosteroids. Indications for antibiotic prescriptions for AAEs are usually not incorporated although the literature shows antibiotics are frequently prescribed. Aim: To investigate the antibiotic prescription rates in AAEs and explore the possible determining factors of those practices. A digital survey was created to determine the antibiotic prescription rates in AAEs and the influencing factors for the prescription practices. The survey was distributed among European academy of allergy and clinical immunology (EAACI) members by mass emailing and through regional/national societies in the Netherlands, Italy, Greece, and Poland. Furthermore, we retrieved local antibiotic prescription rates. In total, 252 participants completed the survey. Respondents stated that there is a lack of guidelines to prescribe antibiotics in AAEs. The median antibiotic prescription rate in this study was 19% [IQR: 0%-40%] and was significantly different between 4 professions: paediatrics 0% [IQR: 0%-37%], pulmonologists 25% [IQR: 10%-50%], general practitioners 25% [IQR: 0%-50%], and allergologists 17% [IQR: 0%-33%]) ($p = 0.046$). Additional diagnostic tests were performed in 71.4% of patients before prescription and the most common antibiotic classes prescribed were macrolides (46.0%) and penicillin (42.9%). Important clinical factors for health care providers to prescribe antibiotics were colorised/purulent sputum, abnormal lung sounds during auscultation, fever, and presence of comorbidities. In 19% of patients with AAEs, antibiotics were prescribed in various classes with a broad range among different subspecialities. This study stresses the urgency to compose evidence-based guidelines to aim for more rational antibiotic prescriptions for AAE.

Immune Mechanisms Underpinning Long COVID: Collegium Internationale Allergologicum Update 2024

Untersmayr E, Venter C, Smith P, Rohrhofer J, Ndwandwe C, Schwarze J, Shannon E, Sokolowska M, Sadlier C, O'Mahony L. Int Arch Allergy Immunol. 2024;185(5):489-502. doi: 10.1159/000535736. Epub 2024 Jan 22.

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection can result in a prolonged multisystem disorder termed long COVID, which may affect up to 10% of people following coronavirus disease 2019 (COVID-19). It is currently unclear why certain individuals do not fully recover following SARS-CoV-2 infection. In this review, we examine immunological mechanisms that may underpin the pathophysiology of long COVID. These mechanisms include an inappropriate immune response to acute SARS-CoV-2 infection, immune cell exhaustion, immune cell metabolic reprogramming, a persistent SARS-CoV-2 reservoir, reactivation of other viruses, inflammatory responses impacting the central nervous system, autoimmunity, microbiome dysbiosis, and dietary factors. Unfortunately, the currently available diagnostic and treatment options for long COVID are inadequate, and more clinical trials are needed that match experimental interventions to underlying immunological mechanisms.

2. Immunometabolism in viral infections, immune tolerance, allergy and asthma

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, microscopy, and Seahorse real-time cell metabolic analysis to examine in vitro cultures of 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).

pulations are impaired ultimately facilitating secondary inflammation but remain poorly studied.

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.

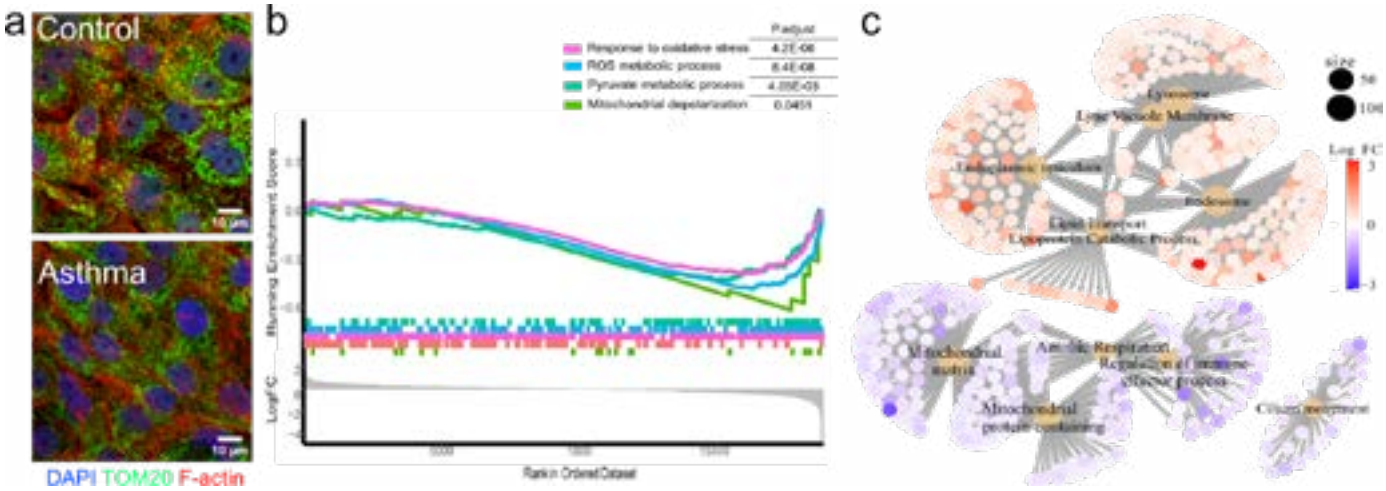


Figure 2. Mitochondrial dysfunction in bronchial epithelium in asthma. (A) Representative confocal microscopy image of TOM20, visualising mitochondria in healthy controls and patients with asthma. (B) GSEA plot showing significantly enriched GO pathways from microarray data of bronchial brushings obtained from patients with asthma (n = 17) and healthy controls (n = 7) (GSE1856580). (C) Cnetplot of key GO GSEA pathways from untargeted proteomics analysis of air-liquid interface-differentiated bronchial epithelial cell lysates, 48 hours post-RV-A16 infection (Control: n = 3, Asthma: n = 3)..

Immunoregulatory role of L-Phenylalanine in 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**. *First co-authors, **Senior co-authors. Under revision.*

Following the completion of a primary immune response and elimination of antigenic challenge, circulating memory CD4+ T effector (Teff) and T regulatory (Treg) cells are established aiding recall responses. However, in allergic and autoimmune diseases, these po-

In this study, we have assessed these key subsets by high-resolution mass spectrometry and observed complete separation of these cell types based on their metabolic profiles which were also enriched in L-phenylalanine (Phe) and L-arginine related metabolites. By combining functional, knockdown, and energy metabolism assessment, we have determined that elevated intracellular Phe specifically promotes glycolysis while simultaneously limiting Oxidative Phosphorylation and proliferation in memory CD4^{+}T cells in an IL4I1-dependent manner. In Th2 cells, Phe exhibited similar effects on energy metabolism along with significantly decreasing expression of key type 2 associated factors involved in Th2 cell function, development, and pathogenicity at mRNA and protein levels (Figure 3). Finally, by incorporating RNA-Sequencing, metabolomics, flow cytometry and proteomics analysis of multiple patient cohorts,

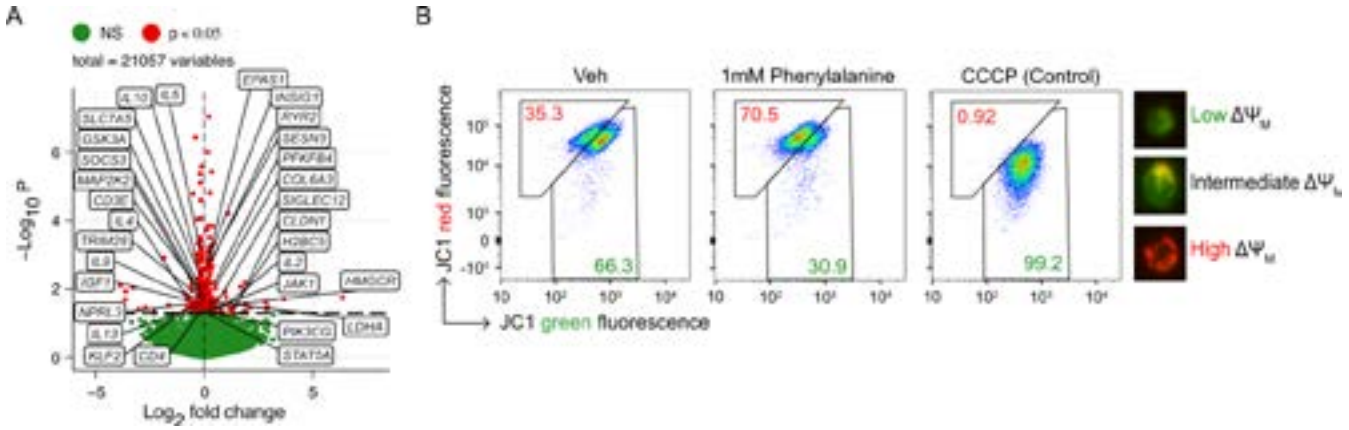


Figure 3. Figure 3. Phenylalanine induced metabolic reprogramming in Th2 cells. (A) Volcano plot representing significant differentially expressed genes in activated Phe-treated (1mM) Th2 cells following 24h treatment (n=5 donors). (B) JC1 staining of Phe- and CCCP-treated CD4+ T cells for the assessment of Phe induced changes in mitochondrial membrane potential.

along with in vitro verification and exploratory assessment we have identified that inefficient import of Phe, due to LAT1 (SLC7A5) dysregulation, and potentially faster intracellular turnover results in a deficit which leads to uncontrolled activation and expansion commonly observed in allergic diseases. Hence, L-phenylalanine plays a critical immunoregulatory role in CD4+ T cell, particularly Th2 cell, biology.

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.

Metabolic pathways in immune senescence and inflammaging: Novel therapeutic strategy for chronic inflammatory lung diseases. An EAACI position paper from the Task Force for Immunopharmacology
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 BCAM, Stellato C. Allergy. 2024 May;79(5):1089-1122. doi: 10.1111/all.15977. Epub 2023 Dec 18.

The accumulation of senescent cells drives inflammaging and increases morbidity of chronic inflammatory lung diseases. Immune responses are built upon dynamic changes in cell metabolism that supply energy and substrates for cell proliferation, differentiation, and activation. Metabolic changes imposed by environmental stress and inflammation on immune cells and tissue microenvironment are thus chiefly involved in the pathophysiology of allergic and other immune-driven diseases. Altered cell metabolism is also a hallmark of cell senescence, a condition characterized by loss of proliferative activity in cells that remain metabolically active. Accelerated senescence can be triggered by acute or chronic stress and inflammatory responses. In contrast, replicative senescence occurs as part of the physiological aging process and has protective roles in cancer surveillance and wound healing. Importantly, cell senescence can also change or hamper response to diverse therapeutic treatments. Understanding the metabolic pathways of senescence in immune and structural cells is therefore critical to detect, prevent, or revert detrimental aspects of senescence-related immunopathology, by developing specific diagnostics and targeted therapies. In this paper, we review the main changes and metabolic alterations occurring in senescent immune cells (macrophages, B cells, T cells). Subsequently, we present the metabolic

footprints described in translational studies in patients with chronic asthma and chronic obstructive pulmonary disease (COPD), and review the ongoing preclinical studies and clinical trials of therapeutic approaches aiming at targeting metabolic pathways to antagonize pathological senescence. Because this is a recently emerging field in allergy and clinical immunology, a better understanding of the metabolic profile of the complex landscape of cell senescence is needed. The progress achieved so far is already providing opportunities for new therapies, as well as for strategies aimed at disease prevention and supporting healthy aging.

Understanding the interplay between psoriatic arthritis and gout: "Psout"

Sherri A, Mortada MM, Makowska J, Sokolowska M, Lewandowska-Polak A. Rheumatol Int. 2024 Dec;44(12):2699-2709. doi: 10.1007/s00296-024-05729-8. Epub 2024 Oct 23.

The interplay between Psoriatic arthritis and Gout is a current diagnostic challenge faced by many physicians and researchers. We aimed at reviewing the coexistence of gout and its features such as hyperuricemia and deposition of monosodium urate crystals in patients with psoriatic arthritis (PsA). We also focused on a brief presentation of the pathophysiology underneath the interplay between PsA and gout, and ultimately on recommendation of approaches for the differential diagnosis. The literature search for this narrative review was conducted using PubMed and Medline and after retrieving and screening the references, articles were selected according to the inclusion and exclusion criteria. Part of the assessed studies reported the coexistence of PsA and gout (Psout) and its association with several clinical outcomes among affected patients. Other studies stressed incidences of misdiagnosis of gout with PsA and vice versa. Additionally, the presence of hyperuricemia in PsA patients could interfere with the patient's characteristics and outcomes of their treatment. Further research on the assessment and clinical course of Psout is required to develop an official protocol for its diagnosis and treatment.

3. Microbiome and nutrition in allergic diseases

A cross talk between microbial metabolites and host immunity: Its relevance for allergic diseases

Losol P, Wolska M, Wypych TP, Yao L, O'Mahony L, Sokolowska M. Clin Transl Allergy. 2024 Feb;14(2):e12339. doi: 10.1002/clt2.12339.

Allergic diseases, including respiratory and food allergies, as well as allergic skin conditions have surged in prevalence in recent decades. In allergic diseases, the gut microbiome is dysbiotic, with reduced diversity of beneficial bacteria and increased abundance of potential pathogens. Research findings suggest that the microbiome, which is highly influenced by environmental and dietary factors, plays a central role in the development, progression, and severity of allergic diseases. The microbiome generates metabolites, which can regulate many of the host's cellular metabolic processes and host immune responses. Our goal is to provide a narrative and comprehensive literature review of the mechanisms through which microbial metabolites regulate host immune function and immune metabolism both in homeostasis and in the context of allergic diseases. describe key microbial metabolites such as short-chain fatty acids, amino acids, bile acids and polyamines, elucidating their mechanisms of action, cellular targets and their roles in regulating metabolism within innate and adaptive immune cells. Furthermore, we characterize the role of bacterial metabolites in the pathogenesis of allergic diseases including allergic asthma, atopic dermatitis and food allergy. Future research efforts should focus on investigating the physiological functions of microbiota-derived metabolites to help develop new diagnostic and therapeutic interventions for allergic diseases.

The influence of lifestyle and environmental factors on host resilience through a homeostatic skin microbiota: An EAACI Task Force Report

Kortekaas Krohn I, Callewaert C, Belasri H, De Pessemier B, Diez Lopez C, Mortz CG, O'Mahony L, Pérez-Gordo M, Sokolowska M, Unger Z, Untersmayr E, Homey B, Gomez-Casado C. Allergy. 2024 Dec;79(12):3269-3284. doi: 10.1111/all.16378. Epub 2024 Nov 1.

Human skin is colonized with skin microbiota that includes commensal bacteria, fungi, arthropods, archaea and viruses. The composition of the microbiota varies at different anatomical locations according to changes in body temperature, pH, humidity/hydration or sebum content. A homeostatic skin microbiota is crucial to maintain epithelial barrier functions, to protect from invading pathogens and to interact with the immune system. Therefore, maintaining homeostasis holds promise to be an achievable goal for microbiome-directed treatment strategies as well as a prophylactic strategy to prevent the development of skin diseases, as dysbiosis or disruption of homeostatic skin microbiota is associated with skin inflammation. A healthy skin microbiome is likely modulated by genetic as well as environmental and lifestyle factors. In this review, we aim to provide a complete overview of the lifestyle and environmental factors that can contribute to maintaining the skin microbiome healthy. Awareness of these factors could be the basis for a prophylactic strategy to prevent the development of skin diseases or to be used as a therapeutic approach.

Nutrition in chronic inflammatory conditions: Bypassing the mucosal block for micronutrients

Roth-Walter F, Berni Canani R, O'Mahony L, Peroni D, Sokolowska M, Vassilopoulou E, Venter C. Allergy. 2024 Feb;79(2):353-383. doi: 10.1111/all.15972. Epub 2023 Dec 12.

Nutritional Immunity is one of the most ancient innate immune responses, during which the body can restrict nutrients availability to pathogens and restricts their uptake by the gut mucosa (mucosal block). Though this can be a beneficial strategy during infection, it also is associated with non-communicable diseases-where the pathogen is missing; leading to increased morbidity and mortality as micronutritional uptake and distribution in the body is hindered. Here, we discuss the acute immune response in respect to nutrients, the opposing nutritional demands of regulatory and inflammatory cells and particularly focus on some nutrients linked with inflammation such as iron, vitamins A, Bs, C, and other antioxidants. We

propose that while the absorption of certain micronutrients is hindered during inflammation, the dietary lymph path remains available. As such, several clinical trials investigated the role of the lymphatic system during protein absorption, following a ketogenic diet and an increased intake of antioxidants, vitamins, and minerals, in reducing inflammation and ameliorating disease.

4. Novel avenues for biomarkers, prevention and treatment of allergy and asthma

European Respiratory Society Research Seminar on Preventing Pediatric Asthma
Grigg J, Barratt B, Bønnelykke K, Custovic A, Ege M, Pasquali C, Palomares O, Shaheen S, Sokolowska M, Vercelli D, Maizels R, von Mutius E. Pediatr Pulmonol . 2025 Jan;60(1):e27401. doi: 10.1002/ppul.27401.

This report is a summary of the presentations given at the European Respiratory Society's Research Seminar on Asthma Prevention. The seminar reviewed both epidemiological and mechanistic studies and concluded that; (i) reducing exposure of pregnant women and children to air pollution will reduce incident asthma, (ii) there are promising data that both fish oil and a component of raw cow's milk prevent asthma, and (iii) modulating trained immunity by either mimicking helminth infection or oral and sublingual bacterial products is a promising area of research

Mesenchymal stromal cells effectively limit house dust mite extract-induced mixed granulocytic lung inflammation
Tynecka M, Janucik A, Tarasik A, Zbikowski A, Niemira M, Kulczynska-Przybik A, Zeller A, Stocker N, Reszec-Gielazyn J, Mroczko B, Kretowski A, Akdis CA, Sokolowska M, Moniuszko M, Eljaszewicz A. Allergy. 2024 Nov;79(11):3157-3161. doi: 10.1111/all.16245. Epub 2024 Jul 19.

The study investigated the effects of adipose tissue-derived mesenchymal stromal cells (MSCs) administered intranasally in a house dust mite (HDM)-induced mixed granulocytic asthma mouse model. MSCs significantly reduced lung inflammation by decreasing eosinophil and neutrophil recruitment and lymphocyte proliferation, as evidenced by transcriptomic analysis. While mucus production remained unchanged, extracellular matrix deposition in the subepithelial area was notably reduced, indicating a partial reversal of remodeling. The findings suggest MSCs could be a promising therapeutic approach for mixed granulocytic asthma by modulating inflammatory pathways.

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.

Digitally-enabled, person-centred care (PCC) in allergen immunotherapy: An ARIA-EAACI Position Paper
Pfaar O, Sousa-Pinto B, Papadopoulos NG, Larenas-Linnemann DE, Ordak M, Torres MJ, Mösges R, Klimek L, Zuberbier T, Matricardi PM, Berger UE, Berger M, Dramburg S, Mahler V, Toppila-Salmi SK, Bergmann KC, Ollert M, Tripodi S, Jutel M, Agache I, Eguiluz-Gracia I, Canonica GW, Akdis CA, Sokolowska M, Sofiev M, Shamji MH, Czarlewski W, Fonseca JA, Bedbrook A, Bousquet J. Allergy. 2024 Aug;79(8):2037-2050. doi: 10.1111/all.16135. Epub 2024 May 3.

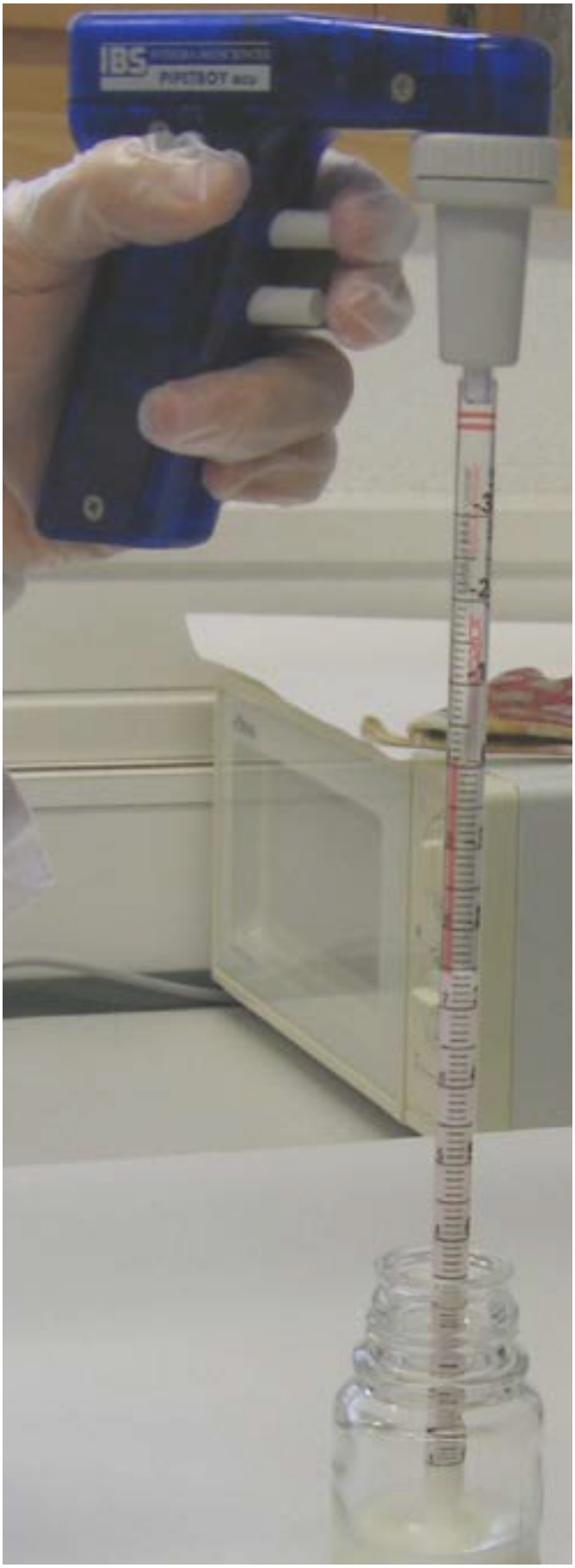
In rhinitis and asthma, several mHealth apps have been developed but only a few have been validated. However, these apps have a high potential for improving person-centred care (PCC), especially in allergen immunotherapy (AIT). They can provide support in AIT initiation by selecting the appropriate patient and allergen shared decision-making. They can also help in (i) the evaluation of (early) efficacy, (ii) early and late stopping rules and (iii) the evaluation of (carried-over) efficacy after cessation of the treatment course. Future perspectives have been formulated in the first report of a joint task force (TF)-Allergic Rhinitis and Its Impact on Asthma (ARIA) and the European Academy of Allergy and Clinical Immunology (EAACI)-on digital biomarkers. The TF on AIT now aims to (i) outline the potential of the clinical applications of mHealth solutions, (ii) express their current limitations, (iii) make proposals regarding further developments for both clinical practice and scientific purpose and (iv) suggest which of the tools might best comply with the purpose of digitally-enabled PCC in AIT.

Type 2 immunity in allergic diseases

Ogulur I, Mitamura Y, Yazici D, Pat Y, Ardicli S, Li M, D'Avino P, Beha C, Babayev H, Zhao B, Zeyneloglu C, Giannelli Viscardi O, Ardicli O, Kiykim A, Garcia-Sanchez A, Lopez JF, Shi LL, Yang M, Schneider SR, Skolnick S, Dhir R, Radzikowska U, Kulkarni AJ, Imam MB, Veen WV, Sokolowska M, Martin-Fontecha M, Palomares O, Nadeau KC, Akdis M, Akdis CA. Cell Mol Immunol. 2025 Mar;22(3):211-242. doi: 10.1038/s41423-025-01261-2. Epub 2025 Feb 17.

Significant advancements have been made in understanding the cellular and molecular mechanisms of type 2 immunity in allergic diseases such as asthma, allergic rhinitis, chronic rhinosinusitis, eosinophilic esophagitis (EoE), food and drug allergies, and atopic dermatitis (AD). Type 2 immunity has evolved to protect against parasitic diseases and toxins, plays a role in the expulsion of parasites and larvae from inner tissues to the lumen and outside the body, maintains microbe-rich skin and mucosal epithelial barriers and counterbalances the type 1 immune response and its destructive effects. During the development of a type 2 immune response, an innate immune response initiates starting from epithelial cells and innate lymphoid cells (ILCs), including dendritic cells and macrophages, and translates to adaptive T and B-cell immunity, particularly IgE antibody production. Eosinophils, mast cells and basophils have effects on effector functions. Cytokines from ILC2s and CD4+ helper type 2 (Th2) cells, CD8 + T cells, and NK-T cells, along with myeloid cells, including IL-4, IL-5, IL-9, and IL-13, initiate and sustain allergic inflammation via T cell cells, eosinophils, and ILC2s; promote IgE class switching; and open the epithelial barrier. Epithelial cell activation, alarmin release and barrier dysfunction are key in the development of not only allergic diseases but also many other systemic diseases. Recent biologics targeting the pathways and effector functions of IL4/IL13, IL-5, and IgE have shown promising results for almost all ages, although some patients with severe allergic diseases do not respond to these therapies, highlighting the unmet need for a more detailed and personalized approach.

Davos, May 2025



EAACI Summer Symposium on Epithelial Cell Biology in Davos, SIAF, July 2024

Dr. Willem van de Veen, PhD

B cell immunology

B cells have a crucial role in IgE-mediated allergies due to their distinctive capability to generate allergen-specific IgE antibodies that bind to high-affinity IgE receptors (FcεRI) on mast cells and basophils, sensitizing them to allergens. Upon subsequent allergen exposure, the crosslinking of FcεRI-bound IgE initiates the release of pro-inflammatory mediators, leading to type I hypersensitivity reactions. In addition to their pro-inflammatory role, B cells, including regulatory B (Breg) cells, can also produce anti-inflammatory cytokines, providing immune regulatory functions. Serological mechanisms of immune regulation by B cells include the increase of IgG4 antibodies in patients undergoing allergen-specific immunotherapy, leading to immune tolerance. Thus, B cells are involved in the development of allergies as well as the development of tolerance to allergens. Our laboratory is interested in investigating various aspects of B cell immunology related to allergies and other immune disorders.

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Dupilumab Treatment Decreases MBC2s, Correlating with Reduced IgE Levels in Pediatric AD

Margot Elise Starrenburg, Laura Buergi, N. Tan Nguyen, Juan F. Lopez-Crespo, Anouk Nouwen, Manal Bel Imam, Nicolette J. T. Arends, Peter J. Caspers, Mübeccel Akdis, Suzanne G. M. A. Pasmans, Willem van de Veen. Journal of Allergy and Clinical Immunology, 2024 Nov;154(5):1333-1338.e4. DOI: 10.1016/j.jaci.2024.06.023

Atopic dermatitis (AD) is a chronic inflammatory skin condition driven by type 2 immunity, which is characterized by elevated levels of IgE. The monoclonal antibody dupilumab, which targets the IL-4 receptor α (IL-4Rα), inhibits IL-4 and IL-13 signaling, both of which play crucial roles in promoting IgE production. Recent findings suggest that type 2 memory B cells (MBC2s), defined as IgG+CD23hiIL-4Rα+ B cells, serve as precursors to IgE-producing cells and are closely correlated with total serum IgE levels. This study aimed to evaluate the impact of dupilumab on MBC2 frequency and total IgE levels in pediatric AD patients and to compare its effects with those of cyclosporine (CsA) and topical corticosteroid therapy.

The study included thirty-six pediatric patients with AD who were randomized into three treatment groups: dupilumab, CsA, and topical corticosteroids. Blood samples were collected at the start of treatment (T0) and after six months (T6). Flow cytometry was used to analyze B-cell populations, while enzyme-linked immunosorbent assay (ELISA) was used to measure total IgE levels.

The findings demonstrated that dupilumab treatment led to a significant reduction in both MBC2 frequency and total IgE levels, an effect not observed in the CsA or topical treatment groups. A strong correlation between MBC2 frequency and total IgE levels was identified, supporting the hypothesis that MBC2s contribute to IgE production. Additionally, dupilumab was found to bind to IL-4Rα on both switched and unswitched B cells, thereby reducing IL-4 and IL-13 signaling, which are essential for IgE class switch recombination (CSR). Although CsA treatment showed a correlation between MBC2 levels and total IgE, it did not significantly reduce overall IgE levels (Figure 1).

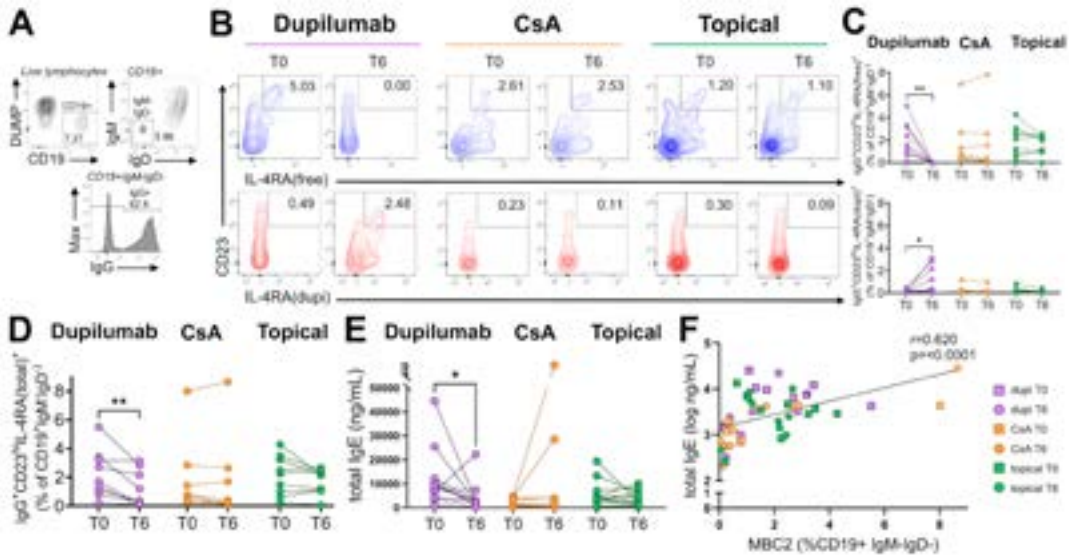

Figure 1. MBC2 frequency, total IgE levels, and correlation. A) Gating strategy for CD19+IgM– IgD– and IgG+ switched B cells. B-D) Density plots and before-after graphs showing the IgG+CD23hiIL-4Rα(free) and G+CD23hiIL-4Rα(dupi)+B-cells as a percentage of CD19+IgM– IgD– B cell population, E) Paired comparisons of plasma total IgE between T0 and T6 in dupilumab, CsA, and topical therapy. F) Correlation of MBC2 frequency in CD19+IgM– IgD– switched B cells and total IgE plasma levels for all patients

These results suggest that systemic IL-4Rα blockade by dupilumab leads to a decrease in circulating MBC2 cells and total IgE levels in pediatric AD patients, revealing a novel mechanism by which the drug influences the atopic immune response. The study highlights the role of IL-4 signaling in MBC2 differentiation and supports the use of dupilumab as a more effective treatment for reducing IgE-mediated atopy in AD compared to CsA or topical corticosteroids. This research provides new insights into the immunological mechanisms of dupilumab and its potential to reshape B-cell memory responses in atopic conditions.

Human Ascaris infection is associated with higher frequencies of IL-10 producing B cells
Zakzuk J, Lopez JF, Akdis C, Caraballo L, Akdis M and van de Veen W. PLOS Neglected Tropical Diseases 18(9): e0012520. 2024. doi: 10.1371/journal.pntd.0012520.

In this study, we investigated the impact of Ascaris lumbricoides infection on immune system responses, with a particular focus on regulatory B cells (Bregs). While the IgE response to A. lumbricoides is well documented, much less is known about how the infection influences cellular immunity, particularly Bregs, which play a role in immunosuppression. Our research was conducted in Santa Catalina, Bolívar, Colombia, a region endemic to soil-transmitted helminthiasis (STH). We analyzed blood samples from eighteen infected individuals and eleven non-infected controls to assess Breg cell populations, related cytokines, and immunoglobulin responses to the parasite's excretory/secretory protein, ABA-1.

Our results demonstrated that infected individuals exhibited significantly higher frequencies of Bregs, with stronger infections correlating with a greater expansion of these regulatory cells. We observed a notable increase in the CD25+CD71+CD73- Breg subset among heavily infected individuals, suggesting a dose-dependent immunosuppressive response. Additionally, we found that infected individuals displayed reduced levels of ABA-1-specific IgG1 and IgE, with IL-10+ B cell frequencies inversely correlating with ABA-1-specific IgE levels. This suggests that A. lumbricoides may suppress antigen-specific humoral responses through increased IL-10-producing Bregs.

Our findings contribute to the growing body of research on helminth-induced immunomodulation, indicating that A. lumbricoides infection can promote immune tolerance by expanding Breg populations. This effect likely serves as an immune evasion strategy for the parasite, allowing it to persist in the host while dampening inflammatory responses. These results also provide insight into broader immunological implications, as the presence of expanded Bregs may affect host susceptibility to other infections, vaccine efficacy, and inflammatory diseases such as allergies and autoimmunity. By understanding the mechanisms through which A. lumbricoides modulates the immune system, we aim to contribute to the development of new therapeutic strategies for controlling excessive immune responses and designing more effective vaccination approaches in helminth-endemic regions.

Optimizing Flow Cytometric Analyses of B-Cell Receptor Immunoglobulin Heavy Chain Isotypes: A Comprehensive Evaluation of Fc Receptor Blocking Strategies and Their Impact on Detection Accuracy
Ozge Ardicli, Margot E. Starrenburg, Juan F. Lopez, Laura Buergi, Cezmi A. Akdis, K. Tayfun Carli, Mübeccel Akdis, Willem van de Veen. Submitted

In this study, we investigated the impact of Fc receptor (FcR) blocking reagents on the accurate detection of B cell receptor (BCR) immunoglobulin heavy chain (IgH) isotypes using flow cytometry. Precise identification of BCR IgH isotypes is crucial for studying B cell maturation and class-switch recombination. However, FcRs on immune cells can interfere with immunophenotyping by binding antibody-based staining reagents. We evaluated different FcR blocking reagents to determine their compatibility with accurate IgH detection and assessed whether a washing step after blocking improved results.

Peripheral blood mononuclear cells (PBMCs) from healthy donors were incubated with five FcR blocking reagents: normal mouse serum, human AB serum, and three commercially available FcR blockers. Cells were stained for IgM, IgD, IgA1, IgA2, and IgG1–4 and analyzed via flow cytometry. We compared detection accuracy between washed and unwashed conditions.

We found that normal mouse serum did not interfere with IgH detection, while human AB serum significantly impaired the detection of IgM+, IgA1-2+, and IgG1-4+ B cells. Proprietary FcR blockers severely disrupted IgG subclass identification, with partial recovery following a washing step. Washing also fully restored IgA1-2+ B cell detection in human AB serum-treated samples. While all tested FcR blockers effectively reduced non-specific antibody binding to monocytes, washing increased background staining.

These findings emphasize the importance of selecting appropriate FcR blocking strategies in flow cytometry. Normal mouse serum emerged as the most suitable FcR blocker, as it did not interfere with IgH detection. While washing improved detection in some cases, it also increased background staining, highlighting the need for tailored blocking approaches to ensure accurate B cell characterization.

Observational study of changes in B cells, Breg cells and amoxicillin-specific immunoglobulins after a type-I hypersensitivity reaction in allergic patients to amoxicillin.
Ruben Fernandez-Santamaria, Maria Salas, Gador Bogas, Cristobalina Mayorga, Mübeccel Akdis, Cezmi Akdis, Maria Jose Torres and Willem van de Veen. In preparation

Immediate drug hypersensitivity reactions (IDHRs) to betalactams, notably triggered by amoxicillin, are on the rise. This study examines the humoral and cellular B cell responses in amoxicillin-allergic patients at various times post-reaction. Using samples from 20 allergic patients and 10 controls, we measured serum levels of specific immunoglobulin E (sIgE) and other immunoglobulins through

tests like RAST and ImmunoCAP, alongside analyzing B cell and B regulatory (Breg) cell frequencies via flow cytometry. Findings reveal early post-reaction phases show high sIgE, indicative of the acute phase, with a notable decrease after six months. Contrarily, specific immunoglobulin G (sIgG) levels drop in the first 12 months then increase, highlighting a potential protective role. Notably, IgG2 and IgG4 levels varied significantly over time, suggesting differential immune responses. The study also found a decrease in certain B cell subsets and regulatory cells immediately following the reaction, with a gradual increase over time. This suggests a complex interplay of humoral and cellular mechanisms in IDHRs to amoxicillin, highlighting the dynamic nature of the immune response over time following an allergic reaction to the drug.

The Role of B Cells and Antibodies in Eosinophilic Esophagitis
Manal Bel Imam, Hang Du, Özge Ardicli, Jenne Meinema, Willem van de Veen. Inflammatory Intestinal Diseases, in press.

In this review, we examine the emerging role of B cells and antibody responses, particularly IgG4, in the pathogenesis of eosinophilic esophagitis (EoE), a chronic, immune-mediated inflammatory disease affecting the esophagus.

Traditionally understood as a Th2-driven disorder characterized by eosinophilic infiltration, recent evidence suggests that B cells and their immunoglobulin products contribute significantly to disease development and progression.
While EoE has been linked to atopic conditions and environmental triggers, the role of B cells and antibodies in sustaining inflammation and tissue remodeling is becoming increasingly recognized

EoE arises from a combination of genetic susceptibility, environmental exposures, and immune dysregulation. A critical component of disease initiation is epithelial barrier dysfunction, which allows antigen penetration and triggers a robust type 2 immune response. This response, driven by cytokines such as IL-4, IL-5, and IL-13, promotes eosinophil recruitment and chronic inflammation. Unlike typical IgE-mediated allergies, EoE appears to involve a shift toward IgG4 production, which is markedly elevated in esophageal tissues of affected patients. This phenomenon, sometimes termed a "modified type 2 response," suggests a distinct immune mechanism in EoE. The presence of IgG4-positive plasma cells and immune complexes in the esophagus points to a role for antibody-mediated inflammation, though whether IgG4 is pathogenic or a bystander remains an open question.

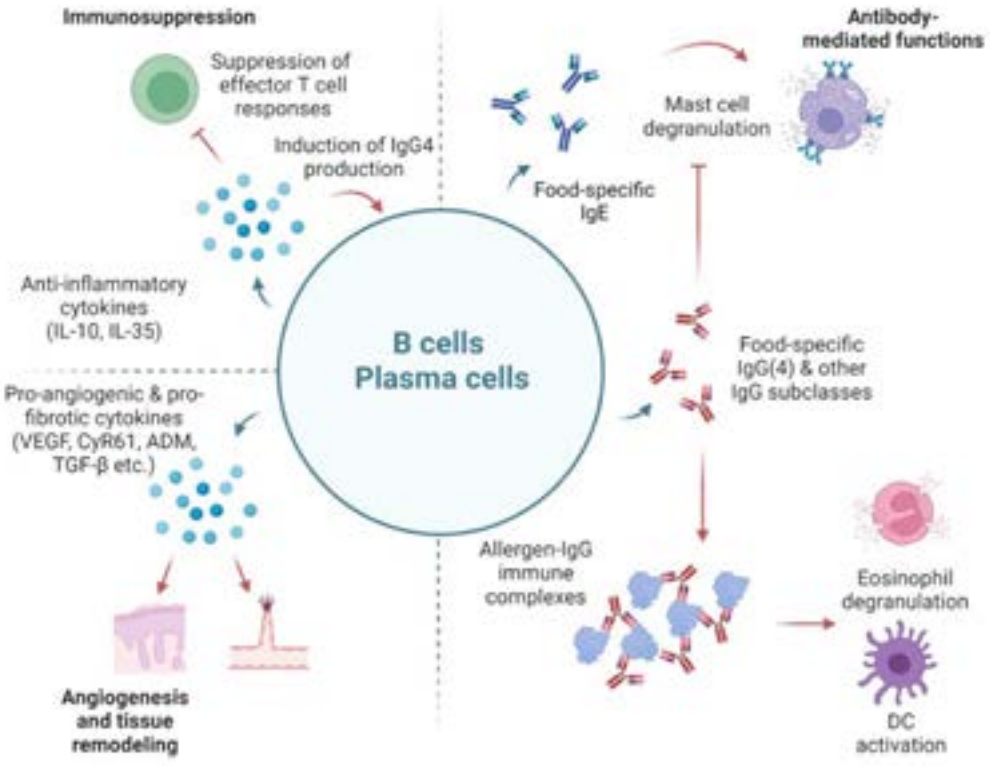

Figure 2. Putative mechanisms of B cell and plasma cell involvement in the immunopathogenesis of EoE. Regulatory B cells, capable of secreting immunosuppressive cytokines, may reduce inflammation by suppressing effector T cell responses. IL-10 can act in an autocrine manner on B cells, shifting antibody production from IgE towards IgG4. In EoE patients, increased frequencies of pro-angiogenic B cells producing various pro-angiogenic and pro-fibrotic cytokines have been identified. These cells may contribute to angiogenesis and tissue remodeling in EoE. The type II cytokine environment in EoE can drive IgE production, especially in response to food antigens, leading to mast cell degranulation. Elevated levels of food-specific IgG4 and potentially other IgG subclasses may inhibit this process. In parallel, food-specific IgG could contribute to inflammation in EoE through immune complex formation, triggering eosinophil degranulation and activating dendritic cells, which subsequently activate allergen-specific T cells. (From Bel Imam et al. Inflammatory Intestinal Diseases, in press

Animal models have provided valuable insights into the mechanisms of EoE, though their lack of IgG4 limits their ability to fully replicate human disease. Clinical studies reinforce the link between esophageal IgG4 levels, eosinophil counts, and histopathologic changes, with some findings suggesting that IgG4 may contribute to tissue remodeling and fibrosis.

While corticosteroids and biologics targeting Th2 cytokines form the mainstay of treatment, they do not directly address the role of B cells.
Dietary elimination remains a cornerstone therapy, but antibody profiling could refine dietary interventions by identifying specific food triggers.
Figure 2 oultines putative roles of B cells and plasma cells in EoE. Future research should further explore the function of B cells and IgG4 in EoE, particularly whether targeting B cells could provide therapeutic benefits. Understanding how B cells interact with other immune components may lead to novel treatments and more personalized management strategies, ultimately improving patient outcomes.

Circulating food allergen-specific antibodies are elevated in eosinophilic esophagitis patients.
Manal Bel Imam, Sayuri Iwasaki, Sophieke Lems, Philipp Schreiner, Mübeccel Akdis, Luc Biedermann, Alex Straumann, Willem van de Veen. Submitted

In this study, we explore the humoral immune response in EoE beyond the well-documented elevation of IgG4, focusing on additional antibody subclasses specific to food allergens. EoE is a chronic inflammatory disorder triggered by dietary antigens, yet its immunopathogenesis remains incompletely understood. While previous research has established a strong association between EoE and increased IgG4 levels, our investigation expands on this by assessing IgG1-3 and IgA1-2 responses to common food allergens such as cow's milk, wheat, and egg.

We analyzed plasma samples from patients with active and inactive EoE, as well as from non-EoE controls, using ELISA to quantify specific antibody levels against key food allergens and their individual proteins, including casein and whey components. Our results confirmed that IgG4 was elevated in EoE, but importantly, we also observed significant increases in other IgG and IgA subclasses. Notably, αS1-casein (Bos d 9) elicited strong IgG and IgA responses, while β-casein (Bos d 11, A1 variant) induced higher IgG2 and IgG4 levels. Among whey proteins, α-lactalbumin (Bos d 4) was associated with an IgG4-dominated response, whereas β-lactoglobulin (Bos d 5) triggered elevations across multiple IgG and IgA subclasses.

Interestingly, despite these systemic antibody elevations, we were unable to isolate food allergen-specific B cells from circulating peripheral blood mononuclear cells, suggesting that antibody production in EoE is likely localized to the inflamed esophageal tissue. This finding aligns with previous studies identifying B lymphocytes and plasma cells in esophageal biopsies.

Our findings challenge the conventional focus on IgG4 as a key marker of EoE and suggest a more complex humoral immune response. This broader antibody profile may have implications for diagnostic strategies and dietary management, potentially guiding more personalized treatment approaches for EoE patients. Future research should explore whether these distinct antibody patterns can serve as biomarkers for disease activity and therapeutic response.

Core Outcome Set for IgE-mediated food allergy clinical trials and observational studies of interventions: International Delphi consensus study 'COMFA'.
Demidova A, Drewitz KP, Kimkool P, Banjanin N, Barzylovich V, Botjes E, Capper I, Castor MAR, Comberiati P, Cook EE, Costa J, Chu DK, Epstein MM, Galvin AD, Giovannini M, Girard F, Golding MA, Greenhawt M, Ierodiakonou D, Jones CJ, Khaleva E, Knibb RC, Macit-Çelebi MS, Mack DP, Mafra I, Marchisotto MJ, Mijakoski D, Nekliudov N, Özdemir C, Patel N, Pazukhina E, Protudjer JLP, Rodríguez Del Rio P, Roomet J, Sammut P, Schoos AM, Schopfer AF, Schultz F, Seylanova N, Skypala I, Sørensen M, Stoleski S, Stylianou E, Upton J, van de Veen W, Genuneit J, Boyle RJ, Apfelbacher C, Munblit D; COMFA Consortium. Allergy. 2024 Mar 3. doi: 10.1111/all.16023.

In this study, we sought to address a critical gap in food allergy research by developing a standardized Core Outcome Set (COS) for IgE-mediated food allergy (FA) clinical trials and observational studies. The inconsistency in reported outcomes across different studies has hindered meaningful comparisons and evidence-based decision-making. To overcome this challenge, the Core Outcome Measures for Food Allergy (COMFA) initiative engaged an international, multidisciplinary group of stakeholders to establish a harmonized set of outcomes that should be consistently measured and reported in FA research.

Our approach involved a comprehensive literature review to identify and categorize relevant outcomes, followed by a two-round online-modified Delphi process and a hybrid consensus meeting. Initially, 39 potential outcomes were identified and refined through iterative discussions, ultimately leading to 13 outcomes being shortlisted for voting. An additional outcome, "quality of life," was added based on participant feedback during the Delphi process. The study engaged 778 participants from 52 countries, representing researchers, healthcare professionals, and individuals with lived experience of FA.

Through the Delphi rounds and consensus discussions, two outcomes—allergic symptoms and quality of life—achieved consensus for inclusion in the final COS. These were deemed essential for evaluating FA interventions, reflecting both clinical significance and patient-centered concerns. While outcomes such as "desensitization" and "remission/sustained unresponsiveness" were recognized as important, they did not meet the predefined inclusion threshold across all stakeholder groups. This suggests that patients and caregivers may perceive these measures differently than researchers

and clinicians, raising concerns about long-term outcomes and symptom recurrence.

Our findings emphasize the need for standardized reporting in FA research. The inclusion of allergic symptoms ensures that trials capture the clinical manifestations of FA, while quality of life accounts for the broader impact of FA on daily living. The next phase of the COMFA initiative will focus on defining optimal measurement tools for these core outcomes to enhance comparability and clinical relevance in FA research.B cell responses and infections

Characterization of B cell responses during immune checkpoint inhibitor treatment in metastatic melanoma
Lacin Cevhertas, Mirjam Fassler , Fiamma Berner , Mübeccel Akdis, Lukas Flatz, Willem van de Veen. In preparation

Immune checkpoint inhibitor (ICI) therapies have gained approval for treating malignant melanoma, yet the response to these treatments varies among patients. This variability underscores the urgent need to discover new biomarkers that can predict how patients will react to ICI therapy and determine their suitability for it. By examining the phenotypes of B cells via surface markers, we can deepen our understanding of B cell immunity in melanoma and how ICIs influence B cells. To this end, we analyzed circulating B cells in healthy individuals and a cohort of 25 metastatic melanoma patients, both before and after they received anti-PD1 and/or anti-CTLA4 monoclonal antibodies, at both early and late stages of their response to the treatment. We found that patients who did not respond to the treatment (n=12) exhibited significant changes in the frequencies of naïve B cells, switched B cells, and IgA+ B cells during their therapy. Notably, B cells from patients who responded to the treatment showed higher expression of the BAFF receptor compared to those from non-responders, right from the baseline and into the early response stage. Furthermore, non-responders had significantly elevated serum levels of BAFF compared to responders at the outset. Our research thus indicates that ICI treatment impacts B cell attributes through the modulation of BAFF receptor expression, and that the soluble protein BAFF might serve as a valuable biomarker for predicting the response of metastatic melanoma patients to ICI therapy.

Davos, May 2025


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PUBLICATIONS

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Schneider S, Satitsuksanoa P, Van de Veen W, Chang I, Babayev H, Yang M, Akdis CA, Nadeau K, Akdis M.Investigation of B-cells in allergy concordant and discordant twins. WIRM 2024, Davos Platz, Switzerland, 13-16 March 2024.

Schneider S, Satitsuksanoa P, Van de Veen W, Chang I, Babayev H, Yang M, Akdis CA, Nadeau K, Akdis M. Investigation of B-cells in allergy concordant and discordant twins. EAACI Congress 2024, Valencia, Spain, 31 Mai -3 Juni 2024.

Schneider S, Satitsuksanoa P, Van de Veen W, Chang I, Babayev H, Yang M, Akdis CA, Nadeau K, Akdis M. Allergy discordant twins do not exhibit differences in gene expression in naive and memory B cells. Graubünden Forscht 2024, Davos Platz, Switzerland, 8-9 November 2024.

Sokolowska M. Novel Mechanisms Controlling Viral and Allergic Inflammation in Asthma and Allergy; KAAACI Seoul International Congress 2024, Seoul, South Korea 10.05.2024

Sokolowska M. Novel Mechanisms of Immunotherapy. The Role of EAACI Research and Outreach Biobank Project; KAAACI Seoul International Congress 2024, Seoul, South Korea 11.05.2024

Van de Veen W. Persistence and Diversity in Allergen-Specific B-Cell Memory: Lessons from Long-term Beekeepers, Novice Beekeepers, and VIT-treated Allergic Patients. EAACI Congress 2024, Valencia, Spain, 31 May - 03 June 2024

Yazici D, Packaged Food Emulsifiers Trigger Pro-Inflammatory Activation and Disruption of the Gut Epithelial Barrier, Graubünden forscht 2024, Davos, Switzerland

Yazici D, Pat Y, Ogulur I, Ardicli S, Simmons S, Almada A, Avena C, Jensen T, Li M, Mitamura Y, Babayev H, Heider A, Dhir R, Akdis M, Nadeau K, Akdis CA. Epithelial Barrier Disruption and Pro-Inflammatory Activation in Gut Epithelial Cells Induced by Food Emulsifiers: Unveiling Epithelitis. World Immune Regulation Meeting XVIII, Davos, Switzerland, 13 – 16 March 2024

Yazici D, Epithelial barrier disruption and pro-inflammatory activation in gut epithelial cells induced by food emulsifiers: Unveiling Epithelitis, World Immune Regulation Meeting 2024 (WIRM), Davos, Switzerland, 15-16 March 2024

Yazici D, Revealing 'Epithelitis': Food Emulsifiers Triggering Pro-Inflammatory Activation and Disruption of the Gut Epithelial Barrier, EAACI Annual Congress 2024, Valencia, Spain, 31 May-3 June 2024

Yazici D, Pat Y, Ogulur I, Ardicli S, Simmons S, Almada A, Avena C, Jensen T, Li M, Mitamura Y, Babayev H, Heider A, Dhir R, Akdis M, Nadeau K, Akdis CA. Revealing 'epithelitis': Food emulsifiers triggering pro-inflammatory activation and disruption of the gut epithelial barrier. EAACI Hybrid Congress 2024 of the European Academy of Allergy and Clinical Immunology, Valencia, Spain, 31 May – 3 June 2024

Yazici D, Packaged Food Emulsifiers Trigger Pro-Inflammatory Activation and Disruption of the Gut Epithelial Barrier, 7th European Congress of Immunology (ECI 2024) - Dublin, 1-4 September 2024

Yazici D, Pat Y, Ogulur I, Ardicli S, Simmons S, Almada A, Avena C, Jensen T, Li M, Mitamura Y, Babayev H, Heider A, Dhir R, Akdis M, Nadeau K, Akdis CA. Packaged Food Emulsifiers Trigger Pro-Inflammatory Activation and Disruption of the Gut Epithelial Barrier. 7th European Conference of Immunology, Dublin, Ireland, 1 – 4 September, 2024

Yazici D, Pat Y, Ogulur I, Ardicli S, Simmons S, Almada A, Avena C, Jensen T, Li M, Mitamura Y, Babayev H, Heider A, Dhir R, Akdis M, Nadeau K, Akdis CA. How common food emulsifiers are breaking down your gut's defense. 9th Conference Graubünden Forscht, Davos, Switzerland, 8 – 9 November 2024

SEMINAR AND CONGRESS TALKS

Akdis CA. The path to become a scientist. Koc University Students, Career Meetings, İstanbul, 8 January 2024, Online Davos, Switzerland

Akdis CA. Uludağ University Carrer Meetings, 10 January 2024, Online Davos, Switzerland

Akdis CA. Epithelial Barrier Theory in Allergic and Autoimmune Diseases Utrecht University, 12 January 2024, The Netherlands,

Akdis CA. Epithelial Barrier Theory, World Economic Forum, MIT Session, Imagination In Action, 17 January 2024, Davos, Switzerland

Akdis CA. Epithelial Barrier Theory: Novel Evidence on Mechanisms of Developmentof Allergic and Autoimmune Diseases. AAAAI Congress, 26 February 2024 Washington DC, USA

Akdis CA. Epithelial Barrier Theory: Novel Evidence on Mechanisms of Developmentof Allergic and Autoimmune Diseases. Harvard University, Chan Institute of Epidemiology, 28 February 2024, Boston, USA

Akdis CA. Epithelial Barrier Theory and Global Health Crisis. Uludag Allergy Meeting, 6 March 2024, Bursa, Turkey

Akdis CA. Epithelial Barrier Theory: Novel Evidence on Mechanisms of Developmentof Allergic and Autoimmune Diseases. Vumedi Seminar 8 March 2024 Davos, Switzerland

Akdis CA. Epithelial Barrier Theory: Novel Evidence on Mechanisms of Developmentof Allergic and Autoimmune Diseases. World Immune Regulation Meeting, 13 March 2024 Davos, Switzerland

Akdis CA. Epithelial Barrier Theory in Allergic and Autoimmune Diseases. Brasil Allergy Congress, 21 March 2024, Online Davos, Switzerland

Akdis CA. Allergic Diseases, Mechanisms, General Overview. Course Zurich University Medical Faculty, 26 March 2024, Davos, Switzerland

Akdis CA. Epithel-barrieren-theorie und Globale gesundheitskrise. German Allergy Congress, 26 March 2024, Online, Davos, Switzerland

Akdis CA. Epithelial Barrier Theory: Novel Evidence on Mechanisms of Development of Allergic and Autoimmune Diseases. Boyman Lab, Akdis Lab Meeting, University Zurich 12 April 2024, Zurich Switzerland

Akdis CA. Epithelial Barrier Theory: Novel Evidence on Mechanisms of Development of Allergic and Autoimmune Diseases. Prof. Hans Uwe Simon's Retirement Ceremony, 12 April 2024, Bern Switzerland

Akdis CA. Epithelial Barrier Theory: Novel Evidence on Mechanisms of Development of Allergic and Autoimmune Diseases. Juna Podcast 15 April 2024, Online, Davos, Switzerland

Akdis CA. Epithelial Barrier Theory and Global Health Crisis. Pediatric Allergy and Asthma Academy CAIAD, 26 April 2024, Cyprus

Akdis CA. Epithelial Barrier Theory: Novel Evidence on Mechanisms of Development of Allergic and Autoimmune Diseases. Mexican Allergy Congress. Plenary Talk. 15 May 2024, Online, Davos, Switzerland

Akdis CA. Allergy Journal Presentations, Executive Committee Meeting, Editorial Board Meeting, Associate Editors Meeting, Reviewers Retreat, 28 May-2 June 2024, Valencia, Spain

Akdis CA. EAACI Journals Session, Allergy. EAACI Congress. 2 June 2024, Valencia Spain

Akdis CA. Epithelial Barrier Theory: Novel Evidence on Mechanisms of Development of Allergic and Autoimmune Diseases. EAACI Congress. Plenary Talk. 3 June 2024, Valencia Spain

Akdis CA. Epithelial Barrier Theory and Mechanisms of Development of Allergic and Autoimmune Diseases. Japanese Dermatology Congress, 6 June 2024, Kyoto, Japan

Akdis CA. Epithelial Barrier Theory. Novartis Japan, 7 June 2024, Kyoto, Japan

Akdis CA. Epithelial Barrier Theory: Novel Evidence on Mechanisms of Development of Allergic and Autoimmune Diseases. Nobel Conference, Stockholm. 13 June 2024, Stockholm, Sweden

Akdis CA. Epithelial Barrier Theory: Novel Evidence on Mechanisms of Development of Allergic and Autoimmune Diseases. Epithelial Congress. 25 July 2024. Alpine Medicine Campus, Davos

Akdis CA. Global Health Crisis and Epithelial Barrier Theory. 1 August 2024, Istanbul, Turkey

Akdis CA. Epithelial Barrier Theory: Novel Evidence on Mechanisms of Development of Allergic and Autoimmune Diseases. European Microplastic Meeting. 28 August 2024, Dublin, Ireland

Akdis CA. Epithelial Barrier Theory: Novel Evidence on Mechanisms of Development of Allergic and Autoimmune Diseases. Cork University. 29 August 2024, Cork, Ireland

Akdis CA. Wiley Session, Experts Guide to Immunology, Allergy-Embracing the Authors and Readers. ECI, European Congress of Immunology. 2 September 2024, Dublin, Ireland

Akdis CA. Epithelial Barrier Theory: Novel Evidence on Mechanisms of Development of Allergic and Autoimmune Diseases. ECI, European Congress of Immunology. 3 September 2024, Dublin, Ireland

Akdis CA. Epithelial Barrier Theory: Mechanisms of Development of Allergic and Autoimmune Diseases. China Allergy Meeting, 3 October 2024, Online, Davos, Switzerland

Akdis CA. Epithelial Barrier Theory: Mechanisms of Development of Allergic and Autoimmune Diseases. Italian Allergy Congress, 4 October 2024, Rome, Italy

Akdis CA. Epithelial Barrier Theory: Mechanisms of Development of Allergic and Autoimmune Diseases. Japanese Allergy Meeting, Kyoto, Japan 15 October 2024, Online, Davos, Switzerland

Akdis CA. Epithelial Barrier Theory: Mechanisms of Development of Allergic and Autoimmune Diseases. BME-364 Course University Zurich 16 October 2024, Davos, Switzerland

Akdis CA. Epithelial Barrier Theory: Mechanisms of Development of Allergic and Autoimmune Diseases. Webinar to APAACI, 30 October 2024, Online, Davos, Switzerland

Akdis CA. Epithelial Barrier Theory: Mechanisms of Development of Allergic and Autoimmune Diseases. Ireland Cork University, PhD Students Lecture, 30 October 2024, Online, Davos, Switzerland

Akdis CA. Epithelial Barrier Theory: Mechanisms of Development of Allergic and Autoimmune Diseases. Imperial College London, Students Lecture, 11 November 2024, Online, Davos, Switzerland

Akdis CA. Epithelial Barrier Theory: Mechanisms of Development of Allergic and Autoimmune Diseases. Egesam Symposium, Izmir Turkey, 15 November 2024, Online, Davos, Switzerland

Akdis CA. Epithelial Barriers and Mechanisms of Development of Allergic and Autoimmune Diseases. Food Allergy and Anaphylaxis Meeting Athens, 27 November 2024, Davos, Switzerland

Akdis CA. Allergic Diseases, Mechanisms, General Overview. Course Zurich University Medical Faculty, 27 November 2024, Davos, Switzerland

Akdis CA. Epithelial Barrier Theory: Novel Evidence on Mechanisms of Development of Allergic and Autoimmune Diseases. Highlights in Allergy Congress 5 December 2024, Oslo, Norway

Akdis CA. Epithelial Barrier Theory and Global Health Crisis. 13 December 2024. Cerrahpaşa University, Istanbul, Online Davos Switzerland

Akdis CA. Indoor CO2 Exposure in Patient Rooms and its Link to Type 2 Response. Asia Pacific Congress of Allergy Asthma and Clinical Immunology (APAAACI) Congress. 13 December 2024. Kuala Lumpur, Malaysia

Akdis CA. Epithelial Barrier Theory: Basis for Suggestions to Public and Policy Makers. Plenary Session. Asia Pacific Congress of Allergy Asthma and Clinical Immunology (APAAACI) Congress. 13 December 2024. Kuala Lumpur, Malaysia

Akdis CA. The Epithelial Barrier Theory: EAACI Session, "68 Diseases, One Health and Veterinary Aspects». Asia Pacific Congress of Allergy Asthma and Clinical Immunology (APAAACI) Congress. 14 December 2024. Kuala Lumpur, Malaysia

Akdis CA. Meet the Expert. „How to write a first-class paper" "How to publish in high journals"
Asia Pacific Congress of Allergy Asthma and Clinical Immunology (APAAACI) Congress. 14 December 2024. Kuala Lumpur, Malaysia

Akdis CA. Evidence Based New Life Style Concepts in the Changing World. 26 December 2024. Fanar, Salallah, Oman

Akdis M. Specific B cell Responses in Allergen Specific Immunotherapy. Gordon Research Conference (GRC), Ventura, LA, USA. 7-12 January 2024

Akdis M. In Vivo Long Term Follow Up of Memory B Cells After Allergen Immunotherapy. American Academy of Allergy, Asthma and Immunology Annual Meeting AAAAI, Washington, DC, USA. 23-26

Seminar and Congress Talks 2024

February, 2024

Akdis M. Breaking Immune tolerance: role of rhinovirus infections in asthma. Harvard T.H. Chan School of Public Health. Boston, USA. 27 February 2024

Akdis M. Specific B cells responses in AIT. Uni Zurich, Clinical Immunology Department. Zurich, 12 April 2024

Akdis M. Breaking Immune tolerance: role of rhinovirus infections in asthma. Europian Academy of allergy and clinical immunology annual congress EAACI, Valencia, Spain. 31 May-3 June 2024

Akdis M. Specific B cells responses in AIT. Eropian Academy of allergy and clinical immunology annual congress EAACI, Valencia, Spain. 31 May-3 June 2024.
Akdis M. Role of B cells in Allergy. Japanese Dermatological Association, Kyoto, Japan. 8 June 2024

Akdis M. Role of Antigen-specific B cells on tolerance induction during AIT. Nobel Conference series at Bergendal estate, Stockholm, Sweden, June 12th-14th 2024

Akdis M. Mechanisms of allergen-specific tolerane; role of B cells. immunology seminar series organized by FInnish Society of Immunology and the InFLAMES flagship program. Finland. 1 October 2024

Akdis M. Allergen-specific immunotherapy. BME 364 module Zurich university. 30 October.2024

Akdis M. Allergen-Specific B Cell in Cow's Milk-OIT. FAAM-EUROBAT, Athens, Greece. 21-23 November 2024

Akdis M. B-Cell Tolerance and Immunotherapy. Asia Pacific Association of Allergy, Asthma and Clinical Immunology (APAAACI), Kuala Lumpur, Malaysia. 12-15 December 2024

Akdis M. Mechanisms that break immune tolerance. Asia Pacific Association of Allergy, Asthma and Clinical Immunology (APAAACI), Kuala Lumpur, Malaysia. 12-15 December 2024

Ardicli O, Starrenburg ME, Buergi L, Akdis CA, Carli KT, Akdis M, van de Veen W. In-depth analysis of FC receptor blocking reagents for enhanced precision in profiling B-cell
receptor immunoglobulin heavy chain isotypes by flow cytometry. EAACI Hybrid Congress 2024 of the European Academy of Allergy and Clinical Immunology, Valencia, Spain, 31 May – 3 June 2024

Ardicli S. Unraveling the impact of Beta-Casomorphin-7 on gut epithelial cells: A comprehensive investigation. World Immune Regulation Meeting XVIII, Davos, Switzerland, 13 – 16 March 2024

Ardicli S. Bovine milk peptide beta-casomorphin-7 alters transcriptomics profiles of gut epithelial cells. EAACI Hybrid Congress 2024 of the European Academy of Allergy and Clinical Immunology, Valencia, Spain, 31 May – 3 June 2024

Ardicli S. Innovative bone materials promote bone formation and simultaneously reduce the
inflammatory response. 9th Conference Graubünden Forscht, Davos, Switzerland, 8 – 9 November 2024

Barletta E. Introduction to Proteomics. Biomedical Data Mining Blockkurs FS 2024 (BME351), Davos, Switzerland, 3-21 June 2024

Barletta E, Fehr DC, Westermann P, Fröhlich K, Brüggen MC, Schmid-Grendelmeier P and Bärenfaller K. Mass spectrometry-based identification of allergen proteins involved in seafood related allergic reactions. WIRM 2024, Davos, Switzerland, 13-16 March 2024

Barletta E, Maurer DJ, Rüegg S, Heider A, Bärenfaller K, Villiger B, Villiger M, Kistler W and Akdis CA. Immune-inflammatory proteomic fingerprinting of elite athletes, amateur athletes and non-sportive controls. WIRM 2024, Davos, Switzerland, 13-16 March 2024

Barletta E, Maurer DJ, Rüegg S, Heider A, Bärenfaller K, Villiger B, Villiger M, Kistler W and Akdis CA. Immune-inflammatory proteomic fingerprinting of elite athletes, amateur athletes and non-sportive controls. Graubünden forscht 2024, Davos, Switzerland, 8-9 November 2024

D'Avino Paolo, DECIPHERING THE EFFECTS OF THE TYPE 2 CYTOKINES IL-4 AND IL-13, AND OF IL- 22 IN EPITHELIAL BARRIER DYSFUNCTION AND SKIN INFLAMMATION IN EX-VIVO HUMAN SKIN, EAACI annual meeting, Valencia, Spain, 31st May-4th June 2024

Du Hang The role of B cells in the pathogenesis of eosinophilic esophagitis MIMretreatCourse, Lenzerheide, Switzerland, 5-7 September 2024

Du Hang. Unveiling Eosinophilic Esophagitis: Our Journey to Understand a Rare Disease.Graubünden forscht, Davos,Switzerland, 8-9.November 2024

Fieten KB. Severe fatigue in uncontrolled asthma: impact of alpine altitude climate rehabilitation and predictive factors for persistent severe fatigue. ERS Congress, Vienna, 07.09.2024 to 11.09.2024

Funch A. Persistent TCR signaling is required for survival of skin-resident allergen specific CD8+ TRM cells. WIRM 2024, Davos, Switzerland 13.03.2024

Funch A. B cell roles in allergic contact dermatitis and in tolerance to contact allergens. Progress report - SIAF 2024, Davos, Switzerland 07.05.2024

Funch A. Wallberg Lecture – Immune mechanism behind local skin reactions to contact allergens. European Society of Contact Dermatitis (ESCD 2024) in Dresden, Germany, 4-7 Sep 2024

Funch A. – B cell roles in allergic contact dermatitis and in tolerance to contact allergens. CARGO meeting 2024 in Copenhagen, Denmark, 4 Oct 2024

Garcia-Sanchez A. Non-nutritive sweeteners cause gut barrier dysfunction with inflammation, oxidative stress, endoplasmic reticulum stress, ribosomal stress and cell death by apoptosis.EAACI Hybrid Congress 2024, Valencia, Spain, 31 May - 03 June 2024
Jardón Parages I. Airway Epithelial Reprogramming in Asthma: Unveiling Viral Infection Dynamics. SIAF Olink Seminar, Zurich, Switzerland, 5 November 2024

Jardón Parages I., Radzikowska U., Huang M., Stocker N., Ding M., Tan G., Heider A., Westermann P., Johnston S., Messner C., Akdis CA., Sokolowska M., Immunometabolic reprogramming of bronchial epithelium in asthma. WIRM, Davos, Switzerland, 14-16 March 2024

Jardón Parages I., Radzikowska U., Huang M., Stocker N., Ding M., Tan G., Heider A., Westermann P., Johnston S., Messner C., Akdis CA., Sokolowska M., Immunometabolic reprogramming of bronchial epithelium in asthma. EAACI Winter School 2024, Zakopane, Poland, 8-11 January 2024

Kulkarni AJ. Metabolomics of circulating human memory CD4+ T effector and T regulatory cells reveals that Phenylalanine is a metabolic checkpoint of pathogenic Th2 cell development. EAACI Immunology Winter School 2024, Zakopane, Poland, 8-11 January 2024

Kulkarni AJ. Metabolic regulation of Th2 cell activity and phenotype by Phenylalanine. World Immune Regulation Meeting XVIII, Davos, Switzerland, 13-16 March 2024

Kulkarni AJ Phenylalanine is a metabolic checkpoint in the development of Th2 cells. EAACI Congress 2024, Valencia, Spain, 31 May – 3 June 2024

Kulkarni AJ. The effect of L-phenylalanine on CD4+ T cell activity. Graubünden forscht 2024, Davos, Switzerland, 8-9 November, 2024

Messner C. B., High-Throughput Proteomics for Biomarker Discovery and Precision Medicine, APMRS-CEEPC 2024, Vienna, Austria, 23.-25. September 2024

Messner C. B., Precision Proteomics (POLAR), Zurich Precision Oncology Symposium, Zurich, Switzerland, 11. March 2024

Mitamura Yasutaka, Unveiling the Impact of Climate Change: Elevated CO2 influences skin epithelial barriers and the development of atopic dermatitis. WIRM Davos, Switzerland 13-16 March 2024

Mitamura Yasutaka,Investigation of cellular infiltrate in atopic dermatitis. EAACI congress 2024, Valencia, Spain, 31 May-3 June 2024

Mitamura Yasutaka, Differential regulation of immune-related genes in the skin of non-responders and allergic individulas following exposure to p-phenylenediamine. EAACI congress 2024 Valencia, Spain, 31 May 2024

Mitamura Yasutaka, Elevated indoor CO2 influence the skin epithelial barrier and the development of atopic dermatitis EAACI Summer Symposium on Epithelial Cell Biology, Davos, Switzerland, 25-26 July 2024

Ogulur I. Gut mucosal responses to environmental toxic substances and food emulsifiers. The EAACI Congress 2024, Valencia, Spain 31 March 3 June 2024

Ogulur I. Gut mucosal responses to environmental toxic substances and food additives. EAACI Summer Symposium on Epithelial Cell Biology, Davos, Switzerland 25-26 July 2024

Ogulur I. Mechanisms of epithelial damage in gut barrier dysfunction: Insights from the epithelial barrier theory. Biomedicine Seminar at University of Zurich, Zurich, Switzerland 6 May 2024

Radzikowska U. Rola inflamasomów nabłonkowych w odpowiedzi immunologicznej na infekcje wirusowe. / The role of epithelial inflammasomes in the immune responses to viral infections. 18th Congress of Polish Society for Fundamental and Clinical Immunology, Bialystok, Poland, 16-18 May 2024

Radzikowska U. Metabolic regulation of epithelial RIG-I signaling in viral exacerbations of asthma. EAACI Immunology Winter School 2024, Zakopane, Poland, 08-11 January 2024

Radzikowska U. Metabolic regulation of epithelial RIG-I signaling in viral exacerbations of asthma. World Immune Regulation Meeting, Davos, Switzerland, 13-16 March 2024

Radzikowska U. Metabolic regulation of epithelial RIG-I signaling in viral exacerbations of asthma. SYIS Annual Symposium 2024, Allschwil, Switzerland, 13-14 June 2024

Radzikowska U. Metabolic regulation of epithelial RIG-I signaling in viral exacerbations of asthma. EAACI Summer Symposium on Epithelial Cell Biology, Davos, Switzerland, 25-26 July 2024

Radzikowska U. Metabolic regulation of epithelial RIG-I signaling in viral exacerbations of asthma. Graubünden Forscht 2024, Davos, Switzerland, 08-09 November 2024

Radzikowska U. Epithelial Immunity in asthma exacerbations and COVID-19. 4th Interdiscipölinary Conference "Jesień Immunologiczna", Rzeszow, Poland, 20-22.11.2024 (online)

Schneider S. Investigation of B-cells in allergy concordant and discordant twins. WIRM 2024, Davos Platz, Switzerland, 13-16 March 2024

Seminar and Congress Talks / Chairs 2024

Schneider S. Investigation of B-cells in allergy concordant and discordant twins. EAACI Congress 2024, Valencia, Spain, 31 Mai -3 Juni 2024

Sokolowska M. Interplay between Immunity and Metabolism in Infection and Type 2 Inflammation. Edinburgh Immunology Group Seminar, Edinburgh, Scotland 29.01.2024

Sokolowska M. Metabolism of allergen-specific immune tolerance. Joint Symposium Swiss Institute for Allergy Research & Department of Immunology. Zurich, Switzerland, 12th April 2024

Sokolowska M. Exposome and Type 2 Inflammatory Diseases. Simposi della Scuola di Specializzazione in Allergologia e Immunologia clinica 2024. 18.04.2024. Milan, Italy

Sokolowska M. Immunity and Metabolism in Infection and Type 2 Inflammation. Training Immunity Workshop OM Pharma, Geneve, Switzerland 23-24 April

Sokolowska M. Novel Mechanisms Controlling Viral and Allergic Inflammation in Asthma and Allergy; KAAACI Seoul International Congress 2024, Seoul, South Korea 10.05.2024

Sokolowska M. Novel Mechanisms of Immunotherapy. The Role of EAACI Research and Outreach Biobank Project; KAAACI Seoul International Congress 2024, Seoul, South Korea 11.05.2024

Sokolowska M. Metabolic control of epithelial inflammation. European Academy of Allergy and Clinical Immunology (EAACI) Congress 2024 31.05.-03.06.2024

Sokolowska M. Mentrorship program (mentor). European Academy of Allergy and Clinical Immunology (EAACI) Congress 2024 31.05.-03.06.2024

Sokolowska M. Crosstalk Between Allergic and Viral-induced Inflammation in Asthma. Federation of Clinical Immunology Societies (FOCIS) Annual Meeting 2024. San Francisco, United States of America, 18.06.2024

Sokolowska M. Viral infections and metabolic regulation of epithelial inflammation. Epithelial Barrier meeting, Davos, Switzerland, 25-26 July 2024

Sokolowska M. Multiomics of interactions between allergens and viruses. Joint International Symposium on Molecular Allergology and the European Rhinallergy Meeting (ISMA-RHINA), Helsinki, Finland 4-5 November

Sokolowska M. Metabolic regulation of pathogenic Th2 cells. Joint International Symposium on Molecular Allergology and the European Rhinallergy Meeting (ISMA-RHINA), Helsinki, Finland 4-5 November

Sokolowska M. Novel Mechanisms Controlling Viral and Allergic Inflammation in Asthma and Allergy. Swiss Society of Allergy and Immunology (SSAI) Annual Congress, Geneva, Switzerland 12-13 September 2024

Sokolowska M. Influence of environment on immunological reactions. XV Polish Allergology Society Meeting, Cracow, Poland 8-10 October 2024

Sokolowska M. Immuno-Metabolic Control of Viral and Allergic Inflammation. Ukrainian School of Molecular Allergology and Immunology. 5-6 December 2024, virtual

Van de Veen W. The role of B cells and antibodies in eosinophilic esophagitis. Symposium on the Occasion of the PhD Defense of Mirelle Kleuskens. Utrecht, Netherlands, 2 January 2024

van de Veen W. In Vivo Long Term Follow Up of Memory B Cells After Allergen Immunotherapy (AIT). 2024 AAAAI Annual Meeting, Washinton DC, USA, 23-26 February 2024

van de Veen W. Novel mechanisms in eosinophilic esophagitis. Joint Symposium Swiss Institute for Allergy Research & Department of Immunology, Zurich, Switzerland, 12 April 2024

van de Veen W. The role of B cells and antibodies in eosinophilic esophagitis. EAACI Congress 2024, Valencia, Spain, 31 May - 03 June 2024

van de Veen W. The Role of B cells in EoE. 2nd Swiss Eosinophilic Esophagitis retreat. Rasses, Switzerland, 22-23 August 2024

van de Veen W. The Role of B cells and Antibodies in Eosinophilic Esophagitis. Symposium: Advances in Immune Regulation. Cartagena, Colombia, 18 September 2024

van de Veen W. The Role of B Cells in Allergy and Immune Tolerance. Symposium: Advances in Immune Regulation. Cartagena, Colombia, 18 September 2024

van de Veen W. The role of B cells and Antibodies in Eosinophilic Esophagitis. Biomedicine Seminar Series. Zurich, Switzerland, 7 October 2024

CHAIRS AT CONGRESSES

Akdis CA. World Immune Regulation Meeting, 13-16 March 2024, Davos Switzerland

Akdis CA. Session 1 Innate Immunity World Immune Regulation Meeting, 13-16 March 2024, Davos Switzerland

Akdis CA. Plenary Session 6. EAACI Congress Valencia 26-28 July 2024, Davos Switzerland

Akdis CA. EAACI Journals Session, EAACI Congress, Valencia 26-28 July 2024, Davos Switzerland

Akdis CA. Allergy Editorial Board Meeting, EAACI Congress, 30 May 2024, Valencia, Spain

Akdis CA. Allergy Associate Editors Meeting, EAACI Congress, 31 May-2 June 2024, Valencia, Spain

Akdis CA. Reviewers Retreat of the Allergy Journal, EAACI Congress, 31 May 2024, Valencia, Spain

Akdis CA. Epithelial Cell Meeting, 26-28 July 2024, Davos Switzerland

Akdis CA. Session 1 Mechanisms of Epithelial Barrier Defects, Epithelial Cell Meeting, 26-28 July 2024, Davos Switzerland

Akdis M. SY25 - Advances in B cell responses in allergy. EAACI Hybrid Congress, Valencia, Spain 31 May – 03 June 2024

Akdis M. YRS1 - Year in Review 1. EAACI Hybrid Congress, Valencia, Spain 31 May – 03 June 2024

Akdis M. PL3 - Nomenclature – EAACI highlights. EAACI Hybrid Congress, Valencia, Spain 31 May – 03 June 2024

Akdis M. EAACI Symposium: The new nomenclature for allergy and hypersensitivity reactions. 7th European Congress of Immunology (ECI), Dublin, Ireland. 1-4 September 2024.

Barletta E. Young Scientists' Satellite 2024. LS2 Annual Meeting 2024, Lausanne, Switzerland, 13-15 February 2024

Barletta E. Regulatory and effector T cells. World Immune RegulationMeeting XVIII (WIRM 2024), Davos, Switzerland, 13-16 March 2024

Mitamura Yasutaka, Session 9 Epithelial barriers. WIRM Davos, Switzerland 15 March 2024

Yasutaka Mitamura. Spatial biology in immunology EAACI Congress 2024, Valencia, Spain, 2 June 2024

Mitamura Yasutaka, Session 8 The epithelial barrier injury and diseases, Davos, Switzerland, 26 July 2024

Ogulur I. Exploring Gut Immunity: The Silent Sentry of Human Health. The EAACI Congress 2024, Valencia, Spain, 31 March - 3 June 2024

Ogulur I. Focus on the epithelial barrier in gut. EEACI Summer Symposium on Epithelial Cell Biology, Davos, Switzerland 25-26 July 2024

Radzikowska U. World Immune Regulation Meeting, Davos, Switzerland, 13-16 March 2024

Radzikowska U. 18th Congress of Polish Society for Fundamental and Clinical Immunology, Bialystok, Poland, 16-18 May 2024

Radzikowska U. SYIS Annual Symposium 2024, Allschwil, Switzerland, 13-14 June 2024

Radzikowska U. EAACI Annual Congress 2024, Valencia, Spain, 31 May - 03 June 2024

Sokolowska M. Keynote 1: Louisa James. The role of tissue resident B cells in the regulation of antibody responses. EAACI Immunology Winterschool, Zakopane, Poland 2024, 08-11 January

Sokolowska M. Poster Session I - Topic 2 Adaptive/innate immunity. EAACI Immunology Winterschool, Zakopane, Poland 2024, 08-11 January

Sokolowska M. Metabolism and Immunity. World Immune Regulation Meeting XVIII Davos, Switzerland, 13-16 March 2024

Sokolowska M. Spatial biology in immunology. European Academy of Allergy and Clinical Immunology (EAACI) Congress 2024 31.05.-03.06.2024

Sokolowska M. Basic immunology/Biomarkers/Immunodeficiency/COVID-19/Infection. European Academy of Allergy and Clinical Immunology (EAACI) Congress 2024 31.05.-03.06.2024

Sokolowska M. Federation of Clinical Immunology Societies (FOCIS)-European Academy of Allergy and Clinical Immunology (EAACI) in partnership with the European Mast Cell and Basophil Research Network (EMBRN) joint symposium. FOCIS Annual Meeting 2024. San Francisco, United States of America, 18.06.2024

Sokolowska M. Periepithelial Inflammation. Epithelial Barrier meeting, Davos, Switzerland, 25-26 July 2024

Sokolowska M. Novel technologies in allergy research. Joint International Symposium on Molecular Allergology and the European Rhinallergy Meeting (ISMA-RHINA), Helsinki, Finland 4-5 November 2024

Van de Veen W. T and B cell memory. World Immune Regulation Meeting XVIII, Davos, Switzerland, 13 - 16 March 2024

Van de Veen W. Infections, pathogenesis and immune responses. World Immune Regulation Meeting XVIII, Davos, Switzerland, 13 - 16 March 2024

Van de Veen W. Immune deficiencies and autoimmunity 4. EAACI Congress 2024, Valencia, Spain, 31 May - 03 June 2024

Yazici D, Transplantation immunology, 7th European Congress of Immunology (ECI 2024) - Dublin, 1-4 September 2024

Yazici D, Respiratory Inflammation, 7th European Congress of Immunology (ECI 2024) - Dublin, 1-4 September 2024

DEGREES

AWARDS and GRANTS

Barletta E. Swiss Young Immunologists Society Award for Best Poster Talk. World Immune RegulationMeeting XVIII (WIRM 2024), Davos, Switzerland, 13-16 March 2024
Barletta E. Aspetar International Young Investigators Award. Pos-

Lectures / Public Seminars 2024

ter Abstract Award 1st Prize. 38th FIMS World Congress of Sports Medicine 2024, Dubai, United Arab Emirates, 24-27 October 2024

D'Avino Paolo, Best poster design, WIRM XVIII in Davos, Switzerland, 13-16th March 2024

D'Avino Paolo, Flash talk award, Spring School 2024: Mechanism of inflamamtion in the skin and other barriers, Hotel Hornbaekhus, Denmark, 15-17 April 2024

D'Avino Paolo, Best Poster Presentation, EAACI annual meeting, in Valencia, Spain, 31st-4th of June 2024

D'Avino Paolo, 1st prize Science day, SIAF in Davos, 19th of December 2024

Funch A. Wallberg Prize. European Society of Contact Dermatitis (ESCD 2024) in Dresden, Germany, 4-7 Sep 2024

Jardón Parages I. Travel Grant Award for Winter School in Zakopane, Poland 8-11 Jan 2024

Jardón Parages I. Congress Grant for EAACI Congress in Valencia, Spain, 31 May-3 June 2024

Mitamura, Yasutaka, EAACI2024 Abstract prize, Valencia, Spain 31 May-3 June 2024

Radzikowska U. SPARK grant number 229131 from SNSF "Understanding LUngs: ImmunoMetabOlism of Succinate in asthma (LUMOS)"

Radzikowska U. EFIS-EJI travel grant. SYIS Annual Symposium 2024, Allschwil, Switzerland, 13-14 June 2024.

Radzikowska U. Best Publication by a Young Researcher 2024 Prize. Swiss Lung Association Research Fund, Baden, Switzerland, May 2024.

Radzikowska U. 24th EAACI Allergopharma Research Award 2024. Europen Academy of Allergy and Clinical Immunology (EAACI), Valencia, Spain, May 2024.

Schneider S. SIAF Science Day Award 3nd Place. SIAF science day in Davos, Switzerland, 19. December 2024

Sokolowska M. Stanford/Elsevier's 2024 Top 2% Scientists

Sokolowska M. 1% Highly Cited Researcher 2024 Clarivate

LECTURES

Akdis CA
University of Zurich 18. + 19.11.2024
University of Zurich 09.+ 10.12.2024
Akdis M.
University Zurich, Biochemistry, Immunology.
Immunology course for Junior Researchers and Master Students

Baerenfalleer K.
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
BME351 Block course "Biomedical Data Mining"; duration: 3 weeks

Mitamura Y.
Block course BME364 10 October-1 November
Atopic dermatitis and type2 inflammation
Allergy and epithelial cells 10 October, Zurich, Switzerland
Laboratory techniques, single cell/spatial RNAseq 25 October

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

Sokolowska M.
University of Zurich: BCH301 Molecular Cell Biology Lectures 02-03.12.2024
BME351 Block course "Biomedical Data Mining"; duration: 3 weeks
BME364 Block course “Mechanisms of allergic disease”, duration 3 weeks

Sokolowska M. Swiss National Science Foundation (SNSF) Weave/Lead Agency grant “Antibodies and microbiota as tools for asthma prevention”

PUBLIC SEMINARS

24.01.2024
Slobodan Tepic, DrSci, CEO, Hepius Biotech AG, Switzerland
“Arginine in arterial vs venous blood in lung inflammation”

12.02.2024
Prof. Wolf-Dietrich Hardt, Professor of Microbiology, ETH Zürich, Institute of Microbiology
“Food components can disrupt colonization resistance against Salmonella gut infections: lessons from mice."

10.03.2024
Dr. Ganesh Pandian Namasivayam
Head and Principle Investigator, Intelligent ChemBioInformatics (INCBI), Kyoto University, Japan
“Flipping the Switch: Exploring Mitochondrial Immunotherapy for Space Travel Adaptation and Asthma Research"

25 - 26 July 2024
EAACI Summer Symposium on Epithelial Cell Biology in Davos, SIAF
Ioana Agache, “Skin epithelial barrier as a biomarker for the asthmatic lung”
Cezmi Akdis, “Restoring the epithelial barrier – key pathways”

Nora Barrett, “The mechanism of airway epithelial remodeling”
Arnaud Bourdin, “Airway epithelial type-2 alarmin profiles”
Jonathan M. Coquet, “Mice with a wild microbiota and strong allergic immune responses”
Alfred Doyle, “Role of Epithelial Barrier Dysfunction in the Pathogenesis of Eosinophilic Esophagitis”
Hamida Hammad, “Mechanisms of the farm dust protective effect”
Marek Jutel, “Effect of biolgicals and current treatments on epithelial cells”
Claudia Mauri, “Exploring Intestinal Permeability: A Novel Approach to Preventing and Treating Autoimmune Diseases”
Hideaki Morita, “Establishment of a novel IL-25-ILC2 dependent mouse model for eosinophilic gastroenteritis”
Jan Hendrik Niess, “Regulatory role of IL-20 subfamily cytokines in oesophageal epithelial barrier function”
Liam O’Mahony, “The interplay between the lung and gut - the important role of the microbiome”
Ali Önder Yildirim, “Regulation of Epithelial Cell Injury in the lung”
Ruby Pawankar, “The One Health concept“
Harald Renz, “Dental Biofilm and Saliva Microbiome and Its Interplay with Pediatric Allergies“
Marek Sanak, “Lipid phenotyping of lung epithelial lining fluid“
Mohamed Shamji, “Innate immune cells and their contribution to diseases“
Milena Sokolowska, “Viral infections and metabolic regulation of lung epithelial barrier”
Martin Steinhoff, “Neuroimmune communication regulating pruritus in atopic dermatitis“
Claudia Traidl-Hoffmann, “Regulation of the epithelial barrier and microbiome by environmental factors”
Andreas Wack, “Repair of airway epithelia requires metabolic rewiring towards fatty acid oxidation“

17.07.2024
Prof. Hugh Sampson, Mount Sinai Hospital, New York, USA, Department of Pediatrics, “Allergenic Epitope Profiling in the Diagnosis & Management of Food Allergy”, “Epicutaneous Immunotherapy for the Treatment of Peanut, Allergy – Basic mechanisms and clinical outcomes"

20.08.2024
PD Dr. med. Johannes Trück, DPhil, Head Division of Allergy, University Children's Hospital Zurich,
Switzerland, "Modern management of food allergy in children"

05.11.2024, SIAF Olink Seminar at the University of Zurich
Adriana Spalwisz, PhD, Olink Technology Presentation
Anja Heider – SIAF, SIAF – Olink Lab Presentation
Prof. Dr. med. Dagmar Simon – Universitätsspital Bern, „Immunological characterization of dupilumab-associated ocular surface disease"
Inés Jardón Parages – SIAF, „Airway Epithelial Reprogramming in Asthma: Unveiling Viral Infection Dynamics“
Kristian Hodén, PhD, Mastering Olink Data: Best Practices and Available Tools
Michal Mateusz Beffinger, PhD – University of Zurich, „Triggering responses in an immune desert: glioblastoma explants meet proteomics“
Milad Ameri – University of Zurich, „Drug Reaction With Eosinophilia and Systemic Symptoms: Deciphering the diversity of systemic Immune profiles“

13.11.2024
Prof. Dr. Dr. med. Silvio Brugger, Department of Infectious Diseases and Hospital Epidemiology, University Hospital Zurich, University of Zurich, Switzerland, “Commensal-pathogen interactions as a source for microbiota-targeted treatments"

SIAF SCIENCE DAY
18.12.2024
Alaa Sherri, "Towards new biomarkers in psoriatic arthritis, the role of anti-inflammatory lipid mediators, and alarmins in the resolution of chronic inflammation"
Philipp Gessner, "Tape Stripping - Peeling back the Layers of Mystery"
Urszula Radzikowska, "It’s Beginning to Look a Lot Like Wheezemas: Asthma’s Metabolic Drama Unfolds as Grinchy Viruses Crash the Party"
Yagiz Pat, "The Historical Perspectives of a Microbial Metabolite"
Hang Du, "The Truth Behind the B Cell Conspirac"
Paolo D’Avino, "Skin Wars: Episode IV- A New Hope"
Stephan Schneider, "A trace of Allergy"
Juan Lopez, "A wild Bee-West story: IgD and the Bee-lligerents"
Bingjie Zhao, "Little Red Bean _ Working hard to compete with MSG and maltol everyday"
Manru Li, "Kung Fu Panda: The Skin Legend – Po's Antioxidant Mission"

Winner of the SIAF Science Day 2024:
Paolo D`Avino

Scientific Posts and Editotial Activities 2024

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
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.

Member of the Human Proteome Organization (HUPO)
Member of Life Science Switzerland, Proteomics and Bioinformatics Section
Member of the Austrian Proteomics and Metabolomics Association (APMA)
Member of the European Academy of Allergy Clinical Immunology (EAACI)
Group leader SIB – Swiss Institute of Bioinformatics
Principal Investigator – Systems Biology PhD program (UZH/ETH)
Principal Investigator – Molecular Life Science PhD program (UZH/ETH)
Reviewer for Nature Communications Reviewer, Nature Biomedical Engineering, ACS omega, Expert review of proteomics"

Mitamura Yasutaka

Editor in Frontiers in Allergy

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

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

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Medical University of Lodz (PL), Prof. J. Makowska, Prof. M. Chalubinski, Dr. F. Styrzynsk

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Nederlands Astmacentrum Davos (CH), Dr. D. Prins, Dr. M. Drijver, Dr. R. Wolters, Dr. K. Fieten

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Semmelweis University, Department of Genetics, Cell- and Immunobiology, Prof. Z. I. Komlósi

Seoul National University, Seoul National University Bundang Hospital, Department of Internal Medicine, Prof. YS Chang, Dr. P Losol

Spital Davos, Dr. W. Kistler, Dr. M. Villiger, Dr. T. Rothe, Dr. A. Speiser

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Swiss Research Institute for Sports Medicine, Davos (CH), Dr. M. Villiger, Dr. W. Kistler

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The Netherlands Cancer Institute, Divison of Cellular Biochemistry, Amsterdam (NL), Prof. P. ten Dijke, Dr. S. Itoh

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The Seventh Affiliated Hospital, Sun Yat-sen University, Prof. Z. Zhiyu

Uludag University of Bursa, Bursa (TR), Prof. H.B. Oral, Prof. F. Budak

Universidad CEU San Pablo, Madrid (SP), Prof. Coral Barbas, Dr. D. Barber, Prof. M.M. Escribese, Dr. A. Villaseñor

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University of Edinburgh, Wellcome Centre for Cell Biology, Dr. G. Kustatscher

Universität Freiburg (D), Institut für Molekulare Medizin und Zellforschung, Prof. O. Schilling

Universität Graz (AT), Deptartement of Pediatrics, Dr. E.M. Varga; Inst. Pharm. Chem., Prof. A. Kungl

Universität Zürich (CH), Biochemical Institute, Prof. M. Grütter, Dr. P Mittl; Clinical Trial Center (CH), PD Dr. G. Senti

Universitätsklinikum Freiburg (DE), COPD & Asthma Research-group (CARG), Abtl. für Pneumologie, PD Dr. M. Idzko

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Universitätsspital Bern (CH), Eggel Lab, Prof. A. Eggel; Kinderklinik, Inselspital, Prof. R. Kraemer, Dr. C. Aebischer-Casaulta, Prof. M.H. Schöni; Universitätsklinik für Rheumatologie, Immunologie und Allergologie, Inselspital, Prof. A. Helbling, Dr. A. Gschwend; Universitätsklinik für Hals-, Nasen- und Ohrenkrankheiten, Kopf- und Halschirurgie, Dr. U. Borner, Dr. S. Negoias, Dr. S.L. Hool

Universitätsspital Zürich (CH); Allergiestation, Dr. C. Cardoso; Abteilung für Klinische Immunologie, Prof. Dr. O. Boyman; Abteilung ENT, PD Dr. D. Holzmann, PD Dr. M. Soyka; Abfteilung Pneumologie, Prof. Dr. M. Kohler, PD Dr. C. Clarenbach; Abteilung Gastroenterologie, Prof. R. Gerhard, Dr. M. Wawrzyniak; Abteilung Kardiologie, Prof. F. Duru, Dr. D. Akdis; Dermatologische Klinik, Prof. R. Dummer, PD Dr. T. Kündig, Prof. Dr. P. Schmid-Grendelmeier, Prof. Dr. M.C. Brüggen, PD Dr. E. Guenova, PD Dr. G. Hofbauer; Swiss EoE

National and international collaborations

Clinics and Research Network, Prof. Dr. A. Straumann, Dr. L. Biedermann, Dr. P. Schreiner; Klinik für Medizinische Onkologie und Hämatologie, Prof. T. Zenz

Universitäts-Kinderspital Zürich (CH), Prof. J. Reichenbach, Prof. R. Lauener, Dr. C. Roduit, Dr. A. Jung

Universitäts-Kinderspital Zürich (CH), Forschungszentrum für das Kind, Klinische Chemie und Biochemie, Dr. P. Wawrzyniak

University of Applied Sciences of the Grisons / Fachhochschule Graubünden (FHGR), DAViS Center (CH), Dr. Heiko Rölke, Marco Schmid, PD Dr. Ralf-Peter Mundani, Keller Thomas, Dr. Yves Staudt

University of Cambridge (UK), Prof. G. Griffith, Dr. C. Ma

University of Cape Town, Department of Dematology (ZA), Assoc Prof. M. Levin, Dr. C. Hlela

University College Cork, Alimentary Pharmabiotic Centre (IE), Prof. Dr. L. O'Mahony, Dr. N. Lunjani, Dr. D. Groeger, Dr. T. Tan

University of Istanbul, Institute of Experimental and Medical Research (TR), Prof. G. Deniz, Prof. Dr. G. Erten, Prof. Dr. U. Kücüksezer, Prof. C. Ozdemir

University of Lausanne, Department of Biochemistry, Lausanne (CH), Prof. M. Thome

University of Natural Resources and Life Sciences, BOKU Wien (AT), Dr. F. Altmann

University of Nottingham (UK), Department of Chemical and Environmental Engineering, Prof. A. Mata

University of Szeged, Department of Dermatology and Allergology, Szeged (HU), Dr. N. Nagy, Prof. L. Kemeny

University of Tartu (EE), Dr. A. Rebane, Prof. P. Peterson, Prof. K. Kingo

Universitätsklinikum St. Pölten, Universitätsklinik für Kinder- und Jugendheilkunde, Prof. T. Eiwegger

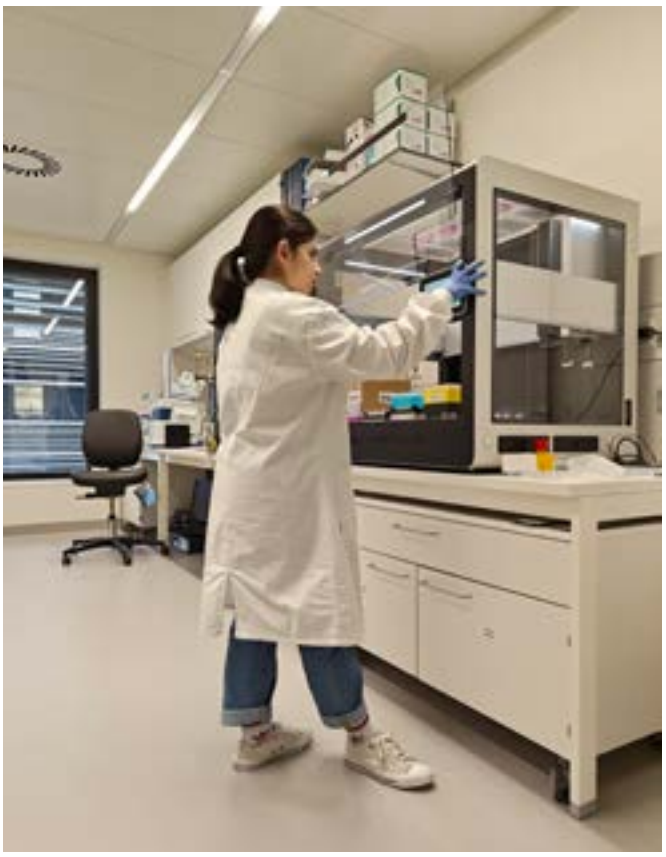
University of Turku, Paediatrics and Adolescent Medicine (FI), Prof. T. Jartti

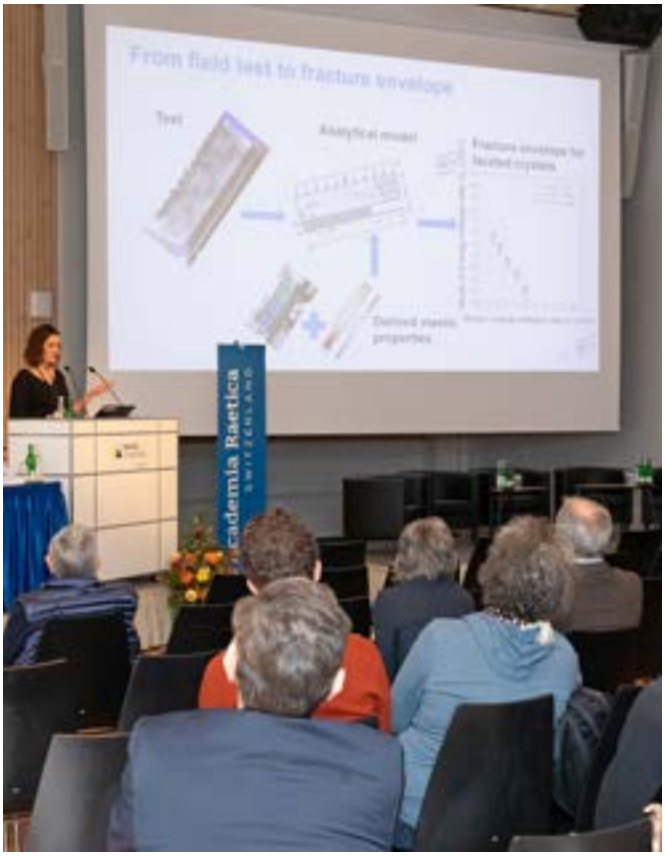
University of Wisconsin-Madison (US), Prof. J. E. Gern

Wroclaw Medical University, Wroclaw (PL), Prof. M. Jutel, Dr. S. Smolinska, Dr. P. Gajdanowicz

Zhongnan Hospital of Wuhan University, Department of Allergology, Dr. Y-D Gao, Dr. M. Ding

EACCI SUMMERSCHOOL 2024 at SIAF





Schweizerisches Institut für Allergie- und Asthmaforschung

Bilanz per 31. Dezember 2024

(inklusive Drittmittel)

	31/12/2024	31/12/2023
	CHF	CHF
AKTIVEN		
Flüssige Mittel	714'607.43	1'410'084.55
Forderungen	235'469.64	426'128.88
Übrige kurzfristige Forderungen	-	73'238.29
Kontokorrent SFI Stiftung	28'542.45	2'302.35
Aktive Rechnungsabgrenzungen	513'274.60	336'209.53
	1'491'894.12	2'247'963.60
PASSIVEN		
Verbindlichkeiten	609'638.78	489'724.78
Kontokorrent PMOD/WRC	5'939.15	-
Übrige kurzfristige Verbindlichkeiten	-	9'051.95
Passive Rechnungsabgrenzungen	760'188.88	1'457'580.72
Rückstellungen	20'221.43	195'700.27
Eigenkapital	95'905.88	95'905.88
	1'491'894.12	2'247'963.60

Schweizerisches Institut für Allergie- und Asthmaforschung

Betriebsrechnung 2024

(inklusive Drittmittel)

	Abschluss 2024	Budget 2024	Abschluss 2023
	CHF	CHF	CHF
ERTRAG			
Beitrag Bund Forschungsgesetz Art. 15	1'292'400.00	1'292'400.00	1'311'700.00
Beitrag Kanton Graubünden	780'000.00	780'000.00	520'000.00
Beitrag Kanton Graubünden Sonderprofessur "Precision-Proteomics-Center"	755'483.28	-	509'546.96
Beitrag Gemeinde Davos	524'560.00	524'560.00	524'560.00
Beitrag Universität Zürich	387'704.70	369'688.28	419'148.90
Beitrag Stiftung SFI	21'618.60	23'000.00	120'000.00
Beitrag Stiftung vormals Bündner Heilstätte Arosa	56'108.50	55'821.00	54'383.50
Beitrag Stiftungen/Drittmittel	-	-	10'000.00
Overheadbeiträge	18'564.00	-	-
Patenterlöse	253'125.78	-	-
Übriger Ertrag	41'879.53	3'000.00	14'334.75
Finanzertrag	-	6'650.00	1'725.95
Ausserordentlicher Ertrag	33'710.98	35'000.00	46'984.93
Auflösung von Rückstellungen	175'478.84	-	-
WIRM-Kongress	228'267.34	256'500.00	225'651.41
Drittmittel	2'980'899.75	2'862'043.00	3'411'906.63
	7'549'801.30	6'208'662.28	7'169'943.03
AUFWAND			
Personalaufwand	3'931'745.63	3'620'630.00	3'434'687.61
Verbrauchsmaterial	1'691'878.17	1'327'320.00	2'077'083.18
Raumaufwand	312'278.74	242'000.00	371'486.79
Unterhalt/Reparaturen/Ersatz	435'103.59	343'000.00	449'709.73
Investitionen	309'018.88	40'000.00	100'963.16
Sachversicherungen/Abgaben	18'106.80	10'000.00	18'465.60
Energie- und Entsorgungsaufwand	166'532.75	187'000.00	209'187.87
Verwaltungsaufwand	171'227.99	105'500.00	105'294.38
Werbeaufwand	17'203.52	65'000.00	20'970.03
Reisespesen	148'957.44		115'615.01
WIRM-Kongress	170'843.46	205'500.00	235'986.73
Übriger Betriebsaufwand	28'311.39	11'000.00	7'503.87
Abschreibungen	105'200.00	105'200.00	105'200.00
Finanzaufwand	473.56	4'000.00	2'514.75
Bildung von Rückstellungen	-	-	-
Ausserordentlicher Aufwand	42'919.38	1'000.00	97.33
Zuweisung/Hertrag Drittmittel	-	-	-
	7'549'801.30	6'267'150.00	7'254'571.38
Ergebnis	-	-58'487.72	-84'628.35
	7'549'801.30	6'208'662.28	7'169'943.03

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