南通大学高层次人才培育计划

培养对象申请表

申 请 人：	崔广为
所在单位：	神经再生重点实验室（与神经科学系、神经再生协同创新中心合署）
申报类别：	A 计划第一档
填表时间：	

南通大学高层次人才工作办公室制

填写说明

一、填写本表前，请认真阅读《南通大学高层次人才支持计划实施办法》和相关通知。

二、申请表中应明确填写清楚申报类别、发展目标、目前已具备的基本条件、未来工作计划等各项内容。

三、表中所涉个人前期业绩成果均应做到真实完整、信息准确、无知识产权纠纷及其他异议。

四、本表请采用 A4 纸双面打印。

一、申请人基本情况

姓　　名	崔广为	性　　别	男	国　　籍	中华人民共和国
出生年月 年龄（周岁）	1985.11 41	政治面貌	群众	党政职务	无
最高学历	博士研究生毕业	最高学位	博士研究生毕业	联系电话	13857183208
现任专业技术职务及任职时间	无				
所在学科一级学科及研究方向	基础医学和药学 免疫学，药理和毒理学，组织工程学				
最高学历毕业学校及毕业时间	京都大学 2015-03-23				
海外学习及工作（含博后）经历	海外学习经历 2006.04-2010.03　大阪公立大学 理学院 生物学（本科） QS 排名 901-950 2010.04-2012.03　京都大学 医学院/病毒研究所 医科学（硕士） QS 排名 57，THE 排名 55 2012.04-2015.03　京都大学 医学院/病毒研究所 医科学（博士） QS 排名 57，THE 排名 55 海外工作经历 2015.04-2016.06　京都大学 病毒研究所 博士后研究员　QS 排名 57，THE 排名 55 2016.07-2023.01　京都大学 病毒与再生医科学研究所 助理教授　QS 排名 57，THE 排名 55 2023.02-2025.11　罗氏(Roche)集团日本中外制药公司 研究员/药物安全性研发负责人				

注：海外经历请注明学习工作高校（科研院所）的世界排名、起止时间及个人职务等情况

二、培育目标及现有基础

申报类别	A 计划第一档	
培育目标	A 计划第一档 基金委 JQ	A 计划第二档
	B 计划	

现有基础	（对照培育目标，简要列举申请人已具备的基础条件，着重标出已取得的标志性成果，500字内） 一、科研项目：1. 日本学术振兴会（JSPS）基金（日本国家级科研基金），编号 17K15721 2017.04－2019.03，20 万，主持；2. 日本学术振兴会（JSPS）基金，编号 19K16687，2019. －2021.03，20 万，主持；3. 日本学术振兴会（JSPS）基金，编号 20K21525，2020.07－2022.0 34 万，参与（排名第二）；4. 日本学术振兴会（JSPS）基金，编号 21K07067，2021.04－2022.1 20 万，主持；5. 罗氏集团内部自研项目，2023.04－2025.11，40 万，主持；6. 罗氏集团内 自研项目，2023.04－2025.11，80 万，主持；7. 罗氏集团内部自研项目，2024.04－2025.11 40 万，主持 二，教学教改项目：无 三、论文、专著：在著名学术期刊上发表 SCI 论文 20 余篇，被论文及专利引用 1100 余次。中以第一作者和通讯作者在 Science Immunol (2022)、PNAS (2023, 2014)、J Immunol (2020) Front Immunol (2024)等国际和日本国内高水平期刊发表论文多篇。研究成果被 Natu Immunology 期刊选为 2022 年度 Research Highlights，并被 Science Japan 关注报道。 四、学术奖励：1. 日本免疫学会 Best Presentation Award，2016；2. 日本武田科学振兴财团 学系研究奖励奖，2017；3. 入选 Nature Immunology 期刊的 Research Highlights，2022 五、荣誉称号：入选江苏特聘教授，2025 六、授权国内与国际发明专利、成果转换情况：罗氏集团内研发成果部分纳入日本专（JP2026040660A），涉及抗体药物研发新技术。构建的高免疫相容性肠道类器官与淋巴结器官模型等实现产业转化，应用于罗氏集团内部抗体药物安全性评估。

三、近五年主要业绩

（一）主要完成或在研的科研项目（作为负责人承担的国家级科研项目可全部列出，一般不超过 5 项）

项目名称	开始时间/到账时间	项目分类	获得资助金额	本人作用	项目级别（纵向）

注：本人作用指负责主持；项目进展情况包括已完成、已完成且成果已转化应用或正在进行等。

（二）近五年主要完成或在研的教学教改项目（作为负责人承担的教学项目可全部列出，一般不超过 5 项）

项目名称	获奖、立项等级	本人排名	获奖、立项时间

（三）近五年主要论文、著作情况

1.主要论文目录（按重要性排序，合计不超过 5 篇）

期刊名称	论文题目	论文级别	发表时间	第一署名单位

2.主要著作目录（按重要性排序，合计不超过 5 本）

书　名	出版社	出版日期

（四）获得主要学术奖励情况（包含科研、教学，按重要性排序，合计不超过 2 项）

获奖项目名称	奖励名称	奖励等级	获奖时间	奖励单位

（五）近五年获得省部级以上荣誉称号情况（按重要性排序，合计不超过 2 项）

荣誉称号	入选时间	颁发部门

（六）授权国内与国际发明专利、成果转换情况（按重要性排序，合计不超过 5 项）

1.授权国内与国际发明专利情况

专利名称	专利号	国别	授权 时间	本人 排名

2.成果转化情况

成果名称	转化 时间	首次受让企业	本人作用 （主要负责或参与）

（七）其他成果

1.其他科研项目

项目名称	开始时间/ 到账时间	项目分类	获得资助金额	项目状态	本人作用	项目级别（纵向）
日本学术振兴会（JSPS）基金（日本国家级科研基金）	2021-04	其他	20	完成	主持	国家级

注：本人作用指负责主持；项目进展情况包括已完成、已完成且成果已转化应用或正在进行等。

2.其他教学教改项目

项目名称	获奖、立项 等级	本人 排名	获奖、立项时间

3.其他论文

期刊 名称	论文题目	论文 级别	发表 时间	学校 署名
Science Immunology	A circulating subset of iNKT cells mediates antitumor and antiviral immunity	三级	2022-10-28	
PNAS	CD45 alleviates airway inflammation and lung fibrosis by limiting expansion and activation of ILC2s	三级	2023-09-05	

Frontiers in Immunology	Insights into the heterogeneity of iNKT cells: tissue-resident and circulating subsets shaped by local microenvironmental cues	四级	2024-02-19	

4.其他著作

书　名	出版社	出版日期

5.其他学术奖励

获奖项目名称	奖励名称	奖励等级	获奖时间	奖励单位
	医学系研究奖励奖		2017-11-01	日本武田科学振兴财团
	科研成果入选年度 Research Highlights		2022-11-21	Nature Immunology 期刊

6.其他授权国内与国际发明专利情况

专利名称	专利号	国别	授权时间	本人排名

8.其他成果转化情况

成果名称	转化时间	首次受让企业	本人作用（主要负责或参与）

四、未来工作计划

突破方向	（对照培育目标及现有条件，分析目标达成中的现实差距及需要突破的方面，500 字内） 申请人已在器官组织特异性免疫微环境、免疫功能异质性调控机制、免疫—内分泌昼夜节律互作以及类器官药物安全性评测体系等方面取得系统性原创成果，并在 Science Immunology 和 PNAS 等国际顶尖期刊发表论文，形成了从基础免疫学到药物研发的完整研究链条。然而，对照人才培育目标，仍存在若干现实差距与亟需突破的方向：一是免疫微环境的组织特异性调控机制虽已初步建立框架，但在细胞谱系精细解析、信号通路动态互作及疾病状态下的功能转化方面仍需更深入的系统性研究；二是免疫系统与神经内分泌系统的昼夜节律性互作虽有理论探索，但缺乏跨尺度、跨模型的整合性证据，亟待依托空间转录组学、能量代谢测定等技术实现机制性突破，以上两项目标在未来五年内形成跨学科原创成果并发表于 Nature，Science 或 Cell 子刊；三是药物安全性评测体系虽已构建高免疫相容性类器官模型，但在免疫反应网络再现及标准化应用方面仍存在瓶颈，需要进一步开发免疫－非免疫器官芯片组与 AI 赋能预测平台，力争推动成果实现产业化应用并获得重大专利授权。申请人将以新技术驱动的免疫调控机制解析为核心，推动类器官/器官芯片在药物研发中的产业化应用，形成具有国际影响力的原创性成果，实现免疫学科发展与生物医药产业升级双重目标。

未来工作计划	（对照培育目标、现有条件及拟突破的方向，拟定未来的主要工作及安排，800 字内） 对照人才培育目标，申请人未来工作将围绕“科学问题原创性突破—科学技术转化—产业应用和产业标准化”三个层面展开，力争在免疫学前沿与药物研发交叉领域形成国际影响力。 一、器官组织特异性免疫微环境研究方面，依托已建立的 IL-15/IL-7 免疫微环境研究框架和组织驻留和外周循环免疫细胞等细胞分类体系，未来将进一步利用空间转录组学、单细胞能量代谢测定和蛋白质组学等新技术，系统绘制器官组织特异性免疫细胞谱系及其信号通路互作图谱。重点突破免疫细胞在疾病、炎症及应激状态下的动态调控机制，明确免疫毒性形成的关键环节。计划在未来五年内在 Nature，Science 或 Cell 子刊发表机制性论文，推动我国在免疫学基础理论上的国际话语权。 二、免疫—神经内分泌系统互作研究方面，针对昼夜节律驱动下免疫功能失衡与疾病易感性问题，拟构建免疫系统与神经内分泌系统的跨尺度互作模型，揭示双向调控机制及功能动态特征。通过特异性敲除小鼠模型和类器官体系，解析免疫毒性与神经内分泌失调的细胞亚群及信号通路作用，力争形成跨学科原创成果并发表于 Nature，Science 或 Cell 子刊，为免疫学与系统生物学交叉研究提供新理论。 三、应用研究与产业转化方面，在药物安全性评测体系方面，突破现有类器官在人源免疫相容体系缺失、免疫反应网络无法再现等瓶颈，构建多类高免疫相容性类器官模型与免疫－非免疫器官芯片组。同时，开发 AI 赋能的药物毒性与免疫原性预测平台，形成下一代药物安全性评测新方法学（NAMs）。目标是在国际期刊发表应用性成果，并推动成果纳入产业化标准，缩短临床前研发周期，降低研发成本，提高研发成功率。 四、人才培养与团队建设方面，依托南通大学神经再生重点实验室，建立跨学科研究团队，吸引免疫学、系统生物学、药物研发等领域青年学者，形成国际化合作网络，推动团队在免疫学与药物研发交叉领域形成持续创新能力。

五、需要学校提供的支持

是否实验类	是	
培育建设经费预算	工作计划	预算（万元）
	新技术驱动的器官组织特异性免疫调控机制与功能异质性解析	30
	新技术驱动的免疫系统-神经内分泌系统昼夜节律性互作机制与功能解析	30
	高免疫相容性类器官/器官芯片驱动的下一代药物安全性评测新方法学（NAMs）开发	40

	合计	100 万元
其他支持需求清单	标准化类器官/器官芯片制备所需仪器设备平台建设	

五、申请人承诺

（对照未来拟开展的工作，列举需要学院及学校提供的其他支持事项的清单）

1. 以上所填信息均真实有效；
2. 如入选学校培育计划，按期完成培育任务，规范使用学校支持经费；
3. 培育目标达成后，本人志愿在本校服务 10 年以上。

申请人签字：_____

_____年_____月_____日

六、二级单位审核推荐意见

1. 申请人填写的内容是否属实？	是
2. 申请人发展目标是否符合学院发展规划？	是
3. 申请人是否具备培育目标的基础条件及培养潜力？	是
4. 申请人对目标达成的现实差距及突破点的分析是否准确？	是
5. 申请人制定的工作计划是否切实可行？	是
6. 申请人对培育建设经费的预算是否合理？	是
7. 学院是否同意推荐申请人申请相关计划？	是
培养期内，学院为申请人提供以下支持： 在研究生名额分配给予倾斜。 优先使用学院科研和教学平台。 提供优质的科研支撑团队。 党政负责人签字： （单位公章） _____年_____月_____日	

七、学校人才工作领导小组意见

经评审，同意_____同志入选_____培育计划。

组长（签章）：

_____年_____月_____日

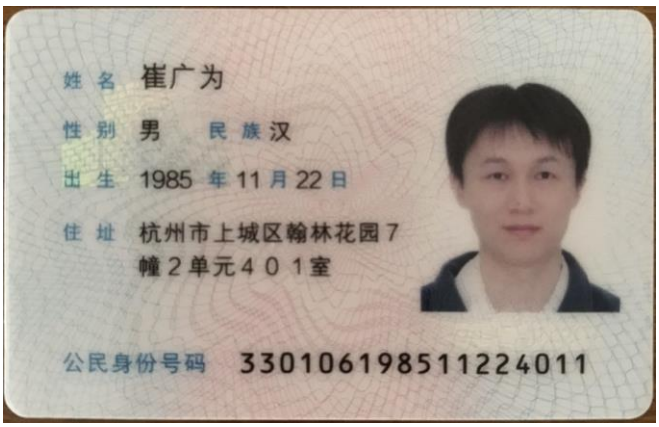
八、学校意见

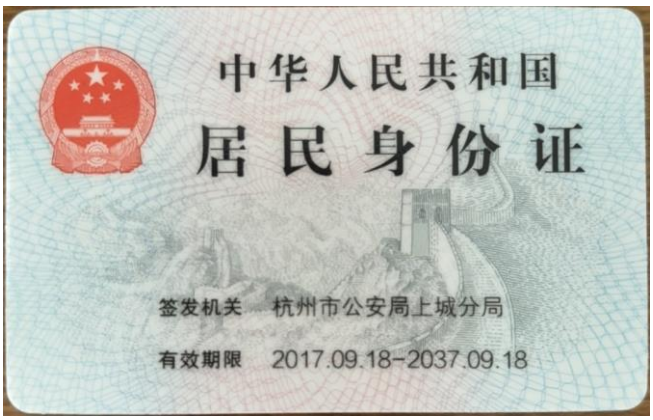
学校公章：　　校长签字：

_____年_____月_____日

申报材料附件

崔广为


姓名 崔广为
性别 男 民族 汉
出生 1985 年 11 月 22 日
住址 杭州市上城区翰林花园 7
幢 2 单元 4 0 1 室
公民身份号码 330106198511224011


中华人民共和国
居民身份证
签发机关 杭州市公安局上城分局
有效期限 2017.09.18-2037.09.18

医科博第５７号

学　位　記

崔　广　为

１９８５年１１月２２日生

本学大学院医学研究科
医科学専攻の博士課程を
修了したので博士（医科学）
の学位を授与する

平成２７年３月２３日

京都大学

KYOTO UNIVERSITY

KYOTO JAPAN

Doctoral Degree No. IKAHAKU 57

This is to certify that

CUI GUANGWEI (born on November 22, 1985)

was conferred the degree of

Doctor of Medical Science

on March 23, 2015, having fulfilled all requirements for

the Department of Medical Science

Doctoral Degree at the Graduate School of Medicine,

Kyoto University, Japan.

March 23, 2015
Kyoto, Japan

Seal of Kyoto University

Juichi Yamagiwa
President
Kyoto University

教育部留学服务中心
Chinese Service Center for Scholarly Exchange

国外学历学位认证书

编号：120250395497

崔广为，男，中国国籍，出生于1985年11月22日。

崔广为在日本京都大学(京都大学)学习，于2015年3月获得该校授予的医科学博士学位，专业领域为医科学。

经核查，京都大学系日本正规高等学校。崔广为所获医科学博士学位表明其具有相应的学历。

教育部留学服务中心

二〇二五年十一月二十八日

注：
1、本认证结果系根据《国（境）外学历学位认证办法》出具。
2、本认证结果中的个人信息系从申请者提供的个人有效身份证件中提取。
3、由于各国（地区）教育制度的差异，认证结果中对申请者专业领域的表述可能与我国《学位授予和人才培养学科目录》及《普通高等学校本科专业目录》等存在差异。

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查询网址：www.cscse.edu.cn

iNKT CELLS

A circulating subset of iNKT cells mediates antitumor and antiviral immunity

Guangwei Cui[1]*, Akihiro Shimba[1,2], Jianshi Jin[3], Taisaku Ogawa[3], Yukiko Muramoto[4], Hitoshi Miyachi[5], Shinya Abe[1], Takuma Asahi[1,6], Shizue Tani-ichi[1,2], Johannes M. Dijkstra[7], Yayoi Iwamoto[8], Kirill Kryukov[9,10], Yuanbo Zhu[1], Daichi Takami[1,11], Takahiro Hara[1], Satsuki Kitano[5], Yan Xu[12], Hajime Morita[8], Moyu Zhang[8], Lynn Zreka[8], Keishi Miyata[13], Takashi Kanaya[14], Shinya Okumura[15], Takashi Ito[15], Etsuro Hatano[15], Yoshimasa Takahashi[16], Hiroshi Watarai[17], Yuichi Oike[13], Tadashi Imanishi[9], Hiroshi Ohno[14], Toshiaki Ohteki[18], Nagahiro Minato[12], Masato Kubo[19,20], Georg A. Holländer[21,22,23], Hideki Ueno[8], Takeshi Noda[4], Katsuyuki Shiroguchi[3], Koichi Ikuta[1]*

Invariant natural killer T (iNKT) cells are a group of innate-like T lymphocytes that recognize lipid antigens. They are supposed to be tissue resident and important for systemic and local immune regulation. To investigate the heterogeneity of iNKT cells, we recharacterized iNKT cells in the thymus and peripheral tissues. iNKT cells in the thymus were divided into three subpopulations by the expression of the natural killer cell receptor CD244 and the chemokine receptor CXCR6 and designated as C0 ($CD244^{-}CXCR6^{-}$), C1 ($CD244^{-}CXCR6^{+}$), or C2 ($CD244^{+}CXCR6^{+}$) iNKT cells. The development and maturation of C2 iNKT cells from C0 iNKT cells strictly depended on IL-15 produced by thymic epithelial cells. C2 iNKT cells expressed high levels of IFN-γ and granzymes and exhibited more NK cell–like features, whereas C1 iNKT cells showed more T cell–like characteristics. C2 iNKT cells were influenced by the microbiome and aging and suppressed the expression of the autoimmune regulator AIRE in the thymus. In peripheral tissues, C2 iNKT cells were circulating that were distinct from conventional tissue-resident C1 iNKT cells. Functionally, C2 iNKT cells protected mice from the tumor metastasis of melanoma cells by enhancing antitumor immunity and promoted antiviral immune responses against influenza virus infection. Furthermore, we identified human $CD244^{+}CXCR6^{+}$ iNKT cells with high cytotoxic properties as a counterpart of mouse C2 iNKT cells. Thus, this study reveals a circulating subset of iNKT cells with NK cell–like properties distinct from conventional tissue-resident iNKT cells.

INTRODUCTION

Invariant natural killer T (iNKT) cells are a group of innate-like T lymphocytes and have diverse functions straddling innate and adaptive immune responses. They are placed at the frontline for protection against cancer and infection and are involved in chronic inflammation. iNKT cells express invariant T cell receptors (TCRs) and recognize lipid antigens presented on CD1d, a nonpolymorphic major histocompatibility complex (MHC) class I–like molecule (*1*, *2*). Upon stimulation by a potent synthetic antigen, α-galactosylceramide (α-GalCer), or inflammatory cytokines, iNKT cells are rapidly activated and produce large amounts of the cytokines interferon-γ (IFN-γ) and interleukin-4 (IL-4), which enhance innate immune responses by activating antigen-presenting cells (APCs) and NK cells (*3*, *4*). Activated iNKT cells induce the maturation of APCs via CD40 ligand and augment adaptive immune responses. They also express Fas ligand and tumor necrosis factor (TNF)–related apoptosis-inducing ligand (TRAIL) and exert cytolytic functions (*2*). iNKT cells are considered to be noncirculating, tissue-resident cells. The regulation and function of iNKT cells differ between tissues, which is important for tissue homeostasis and immunity (*5*, *6*). However, how iNKT cells acquire their heterogeneous homeostasis and function remains unclear.

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CD45 alleviates airway inflammation and lung fibrosis by limiting expansion and activation of ILC2s

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Group 2 innate lymphoid cells (ILC2s) are critical for the immune response against parasite infection and tissue homeostasis and involved in the pathogenesis of allergy and inflammatory diseases. Although multiple molecules positively regulating ILC2 development and activation have been extensively investigated, the factors limiting their population size and response remain poorly studied. Here, we found that CD45, a membrane-bound tyrosine phosphatase essential for T cell development, negatively regulated ILC2s in a cell-intrinsic manner. ILC2s in CD45-deficient mice exhibited enhanced proliferation and maturation in the bone marrow and hyperactivated phenotypes in the lung with high glycolytic capacity. Furthermore, CD45 signaling suppressed the type 2 inflammatory response by lung ILC2s and alleviated airway inflammation and pulmonary fibrosis. Finally, the interaction with galectin-9 influenced CD45 signaling in ILC2s. These results demonstrate that CD45 is a cell-intrinsic negative regulator of ILC2s and prevents lung inflammation and fibrosis via ILC2s.

innate lymphoid cell | airway inflammation | fibrosis | CD45 | metabolism

Significance

ILC2s trigger the pathogenesis of allergic airway inflammation and pulmonary fibrosis. However, the regulatory mechanism limiting ILC2 population size and inflammatory responses remain poorly studied. This study identified CD45 as a regulatory factor of ILC2s in the steady state and inflammations. ILC2s in the CD45-deficient mice exhibited enhanced proliferation and maturation in the bone marrow and hyperactivated phenotypes in the lung with elevated glycolytic metabolism. Functionally, CD45 signaling suppressed the lung ILC2s and was involved in the alleviation of airway inflammation and pulmonary fibrosis. Our study highlights that CD45 functions as a unique regulator of ILC2s for suppressing excessive type 2 inflammatory responses.

Group 2 innate lymphoid cells (ILC2s) are critical for response against parasite infections and tissue homeostasis (1, 2). ILC2s are tissue-resident lymphocytes enriched in mucosal tissues such as the lung and intestine (3). GATA3, the master transcription factor of ILC2s and Th2 cells, is essential for ILC2s development and function (4). In addition, developing ILC2s express transcriptional factors such as Id2, TCF-1, RORα, and Gfi1, which are important for T cell differentiation (2, 5–7). In the adult bone marrow, ILC2s develop from common lymphoid progenitors (CLPs), followed by common helper-like ILC progenitors (CHILPs), and finally differentiate into ILC2 precursors (ILC2Ps) and mature ILC2s (mILC2s) (8).

Multiple molecules that positively control the development and activation of ILC2s have been investigated. First, ST2, a subunit of the IL-33R, is expressed by almost all tissue ILC2s. IL-33, a cytokine released from endothelial cells, epithelial cells, and preadipocytes, binds to ST2, promotes the proliferation of ILC2s, and elevates the production of type 2 cytokines such as IL-5 and IL-13 (1). Second, IL-17RB, a component of the IL-25R, is highly expressed on ILC2s in the intestine. Upon IL-25 stimulation, some ILC2s are activated and become inflammatory ILC2s, which produce IL-17 in addition to type 2 cytokines (9, 10). Third, ILC2s express cytokine receptors such as CD25 (IL-2Rα), CD127 (IL-7Rα), and thymic stromal lymphopoietin receptor (TSLPR), which transmit signals for the homeostasis and activation of ILC2s (2, 11–13). Finally, ILC2s express several costimulatory molecules critical for T cell immune response, such as inducible T cell costimulator (ICOS), tumor necrosis factor receptor 2 (TNFR2), and glucocorticoid-induced TNFR-related protein (GITR), which enhance the effector functions of ILC2s (14–16).

Although the population size of ILC2s is controlled in the steady state, ILC2s expand after activation and exert critical functions at the initiation and amplification phases of type 2 inflammation. Conversely, excessive inflammatory reactions of ILC2s trigger the pathogenesis of asthma, pulmonary fibrosis, and acute lung injury. Asthma is induced and exacerbated by the type 2 cytokines ILC2s and Th2 cells produce. IL-5 promotes eosinophil activation and recruitment to the lung, and IL-13 induces goblet cell hyperplasia and mucus production (17). ILC2s increase in number in asthma patients and exhibit activated phenotypes (18). Furthermore, type 2 immunity plays an essential role in idiopathic pulmonary fibrosis. The type 2 cytokines from ILC2s act on lung fibroblasts to elevate collagen production and promote fibrosis (19, 20). Thus, elucidating the regulatory mechanisms of ILC2s in the steady state and inflammation is vital to prevent and treat these respiratory diseases.

Several reports have shown that some molecules suppressing T cell activation, such as IFNR, IL-27R, and programmed cell death 1 (PD-1), are induced in ILC2s upon activation or maturation and suppress the activated ILC2s (21–23). Both type 1 and type 2

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Insights into the heterogeneity of iNKT cells: tissue-resident and circulating subsets shaped by local microenvironmental cues

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Invariant natural killer T (iNKT) cells are a distinct subpopulation of innate-like T lymphocytes. They are characterized by semi-invariant T cell receptors (TCRs) that recognize both self and foreign lipid antigens presented by CD1d, a non-polymorphic MHC class I-like molecule. iNKT cells play a critical role in stimulating innate and adaptive immune responses, providing an effective defense against infections and cancers, while also contributing to chronic inflammation. The functions of iNKT cells are specific to their location, ranging from lymphoid to non-lymphoid tissues, such as the thymus, lung, liver, intestine, and adipose tissue. This review aims to provide insights into the heterogeneity of development and function in iNKT cells. First, we will review the expression of master transcription factors that define subsets of iNKT cells and their production of effector molecules such as cytokines and granzymes. In this article, we describe the gene expression profiles contributing to the kinetics, distribution, and cytotoxicity of iNKT cells across different tissue types. We also review the impact of cytokine production in distinct immune microenvironments on iNKT cell heterogeneity, highlighting a recently identified circulating iNKT cell subset. Additionally, we explore the potential of exploiting iNKT cell heterogeneity to create potent immunotherapies for human cancers in the future.

KEYWORDS

iNKT cell, heterogeneity, immune microenvironment, IL-15, immunotherapy

Introduction

Invariant natural killer T (iNKT) cells are a subset of T lymphocytes with innate-like properties that detect self or foreign lipid antigens presented on non-polymorphic major histocompatibility complex (MHC) class I-like molecule CD1d (1–3). They express semi-invariant T cell receptors (TCRs) encoded by Vα14-Jα18 in mice and Vα24-Jα18 in humans, paired with a limited number of TCRβ chains Vβ8.2, Vβ7, or Vβ2 in mice and Vβ11 in humans (4). iNKT cells become activated rapidly at the onset of immune responses

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