세계선도 중점학과 육성사업
결과보고서

소 속	분자의학 및 바이오제약 학과		
직 급	석사과정	성명	곽신영
승인여행기간	2018 . 10 . 31 ~ 2018 . 11 . 03		
여 행 기 간	2018 . 10 . 31 ~ 2018 . 11 . 03		
여행국(도시명)	대한민국 (서울)		
여 행 목 적	10th International Conference on Heme Oxygenase 2018 학회 참석		
주요 방문일정	▪ 대한민국 서울, 학회 청강 및 행사 참석 (10. 31 - 11. 03) ▪ Heme oxygenase 관련 강연 (서울대학교 중강당) ▪ Welcome reception (서울대학교 호암 교수회관) ▪ Gala dinner (서울 세빛섬)		
활 동 내 용 (보고서요약)	▪ 10. 31 - 11. 03 학회 참석 ▪ 10. 31 Welcome reception ▪ 11. 01 - 11. 02 포스터 발표 (Heme oxygenase-1 mediates engulfment of apoptotic colonic epithelial cells by macrophages during the resolution of dextran sodium sulfate-induced murine colitis) ▪ 11. 02 Gala dinner		
제 출 인	곽신영		
첨 부	1. 출입국사실증명서 1부. 2. (사전 미제출시)논문초록 1부. 3. (사전 미제출시)국제점검기준표 1부. 끝.		

국제 학술대회 점검 기준표

소속 학과	분자의학 및 바이오제약학과		
학회일정	2018. 10. 31- 11. 03	사업명	세계선도 중점학과 육성사업
학술회의명	The 10th international conference on heme oxygenase 2018	학회지(장소)	서울

1. 주관기관 : GCRC

2. 총 참여 국가(발표자의 소속국가) : 18개국 (Korea, British, Austria, Pakistan, German, French, Thai, Taiwan, United States, Czech, Italian, Valášková, China, Indian, Japan, Poland, Portugal, Australia)

3. 총 발표 논문 수

구두발표 수	포스터 발표 수	총 발표 논문 수
54	56	110

4. 구두발표 논문 중 외국인 발표자 비율

한국인 비율 (건수)	외국인 비율 (건수)	전체 논문 비율 (건수)
24.07 % (13)	75.93 % (41)	100 % (54)

*외국인 비율: 구두발표논문의 제1저자의 소속기관을 기준으로 산정.
 - 제1저자가 논문을 제출할 때의 소속기관을 기준으로 함. 외국기관 소속 한국 국적의 사람일 경우 외국인 논문비율에 포함. 한국 기관에 소속된 외국인은 외국인논문비율에 포함하지 않음
 - 제1저자의 구분이 없는 경우, 저자 수의 50% 이상이 되는 저자 소속 기관의 국적으로 판정

5. 관련규정 : 과학기술(기초, 융복합)분야
 - 4개국(대한민국 포함)이상 참여
 - 총 구두발표 논문이 20건 이상
 - 구두발표의 논문 발표자 중 외국기관 소속 외국인이 50% 이상

결재	지도교수	

P-41

Heme oxygenase-1 mediates engulfment of apoptotic colonic epithelial cells by macrophages during the resolution of dextran sodium sulfate-induced murine colitis

Shin-Young Gwak[1,2], Seung Hyeon Kim[2], Ishrat Aklima Muna[2], Young-Joon Surh[1,2]

[1]Department of Molecular Medicine and Biopharmaceutical Sciences, Graduate School of Convergence Science and Technology and [2]Tumor Microenvironment Global Core Research Center, College of Pharmacy, Seoul National University, Seoul, South Korea

An injured and infected tissue goes through several steps to restore homeostasis during the resolution of inflammation. One of them, clearance of dead cells including apoptotic immune cells, termed 'efferocytosis', could be an essential step that changes the inflammatory environment from pro-inflammatory to pro-resolving state. A majority part of clearance of the dead cells is accomplished by phagocytes. After efferocytosis, macrophages are polarized to the M2 phenotype, thereby transmiting immunosuppressive signaling. However, the molecular mechanisms underlying macrophage polarization after efferocytosis remains largely unraveled. In the present study, bone marrow derived macrophages (BMDM) treated with apoptotic colonic epithelial cells showed significantly increased heme oxygenase-1 (HO-1) and nuclear factor-like 2 (Nrf2) expression. The percentage of $CD206^{+}$M2 like macrophage, a key factor in the resolution of inflammation, was also increased. Interestingly, genetically knocking out or pharmacologic inhibition of HO-1 with zinc protoporphyin IX (ZnPP) abrogated the upregulation of CD206 in macrophages treated with apoptotic colonic epithelial cells. Dextran sulfate sodium (DSS) is a chemical reagent inducing colitis by damaging colonic epithelium. We found that the resolution of colitis started 3 days after termination of DSS treatment for 7 days which was accompanied by upregulation of HO-1. ZnPP treatment hampered the normalization of the numbers of infiltrated total leukocytes, granulocytes and myeloid lineages during the resolution of DSS-induced murine colitis. The proportion of $CD206^{+}$M2 like macrophage was also decreased. Furthermore, the survival rate decreased dramatically in mice treated with ZnPP. These results suggest that the resolution of DSS-induced colitis is mediated by HO-1 inactivation. In conclusions, HO-1 induced during the efferocytosis plays a key role in the resolution of DSS-induced colitis by modulating the polarization of macrophages.

Keywords: Acute colitis; efferocytosis; M2 macrophages; resolution of inflammation