

Rheumatoid Arthritis

류마티스 연구를 위한 웅비메디텍 제품 가이드

RA 관련 연구 분야

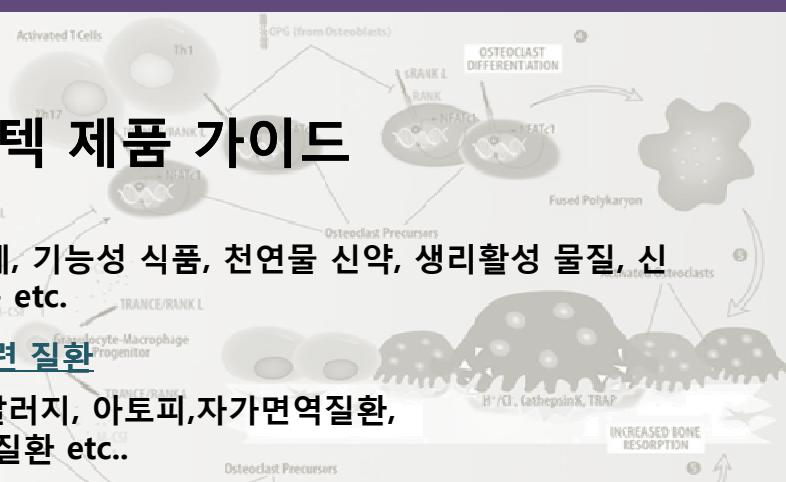
Rheumatoid Arthritis



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연관 분야 : Inflammation 관련 질환

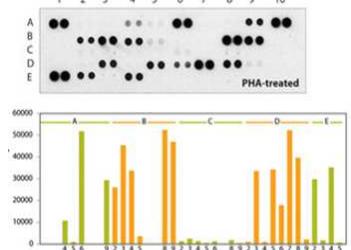
퇴행성 관절염, 천식, 비염, 알러지, 아토피, 자가면역질환, 염증성 장질환, 각종 대사성질환 etc..



1. 초기 연구 (pro-inflammatory cytokine, 염증유발인자, 선도 물질 screening)

nitric oxide	염증유발인자	R&D	KGE001
PGE2	염증유발인자	R&D	KGE004B
iNOS	염증유발인자	R&D	KCB9502
COX-2	염증유발인자	R&D	KCB4198/ DYC4198
thromboxane	염증유발인자	R&D	KGE011
cAMP	염증유발인자	R&D	KGE002B
Hyaluronan	관절구성물질	R&D	DY3614
Hyaluronan	관절구성물질	R&D	GLR001~004
SOD	항산화	R&D	7500-500-k
MPO	항산화	OXIS	21013
LactoF	항산화	OXIS	21015
AOP	항산화	OXIS	21053

SPECIAL TOOL !
Proteom Profiler
Human cytokine array



소량의 sample로
36가지 cytokine을 한번에 screening!!

2. RA 진행 과정 별 (파골 과정)

진행 과정	관련 marker
T cell이 관절 부위로 침투	Angiogenesis 관련 ELISA _ VEGF/FGF/PDGF etc. (R&D) Angiogenesis multiplex & Growth factor multiplex (R&D)
각종 염증 유발인자 및 단백질 분해효소 분비	Inflammation (Cytokine, TIMP)/ MMP 관련 ELISA & Multiplex (R&D) Collagen 관련 – CICP, helical peptide, PYD, DPD (Quidel)
염증 관련 cytokine 분비	Cytokine ELISA/ Array / Multiplex(F-MAP, Mosaic ELISA) (R&D)
T cell로 부터 RANKL/TRANCE 발현	RNKL ELISA (R&D / Quidel / Biovendor/ Bio medica/Immundiagnostik) OPG ELISA (R&D / Quidel/ Biovendor/ Bio medica/Immundiagnostik)
파골 세포 분화 & 파골 진행	Cathepsin K / DKK-1/ Sclerostin (Biomedica) BAP/ YKL-40 / Osteocalcin / TRAP5b / Sclerostin (Quidel) Osteonectin / BAFF / BSP/ vitaminD (Immundiagnostik)

3. 기타 류마티스 관련 연구 (바이오 의약품, 초기 진단)

TNF- α receptor ELISA (Immundiagnostik)

TNF α -Blocker-ADA (anti-drug-antib./Remicade),
TNF α -Blocker-ADA (anti-drug-antib/Humira)
TNF α -Blocker-ADA (anti-drug-antib/Enbrel)
TNF α -Blocker-Monitoring (drug-leve/Remicade)
TNF α -Blocker-Monitoring (drug-level/Humira)

IL-6 receptor

hIL-6 receptor ELISA (R&D)
hIL-6 ELISA (R&D)

P38

P38 alpha/ delta/ gamma ELISA (R&D)
P38 MAP kinase (R&D)

CD40 (TRAP) _ h CD40 ELISA (R&D)
Osteoactivin _ h Osteoactivin (R&D)

CRP

hCRP ELISA (R&D)

NF- κ B

NF- κ B ExactaChip (R&D)

PDGF-BB

PDGF-BB ELISA (R&D)

제품별 상세한 정보는 웅비 학술 마케팅 팀 (031-776-3300)으로 전화 주세요



Cross Talk Between the Immune & Skeletal Systems

Bone remodeling is a normal, homeostatic process that is mediated by bone-forming osteoblasts and bone-resorbing osteoclasts. Factors that alter the activities of these cells can disrupt bone stability and cause an increase or a decrease in bone mass. Rheumatoid arthritis is a chronic inflammatory disorder characterized by excessive bone loss. In this disorder, T cells infiltrate tissues in the joints **①** and release pro-inflammatory cytokines including IL-17 and TNF- α **②**. These cytokines stimulate the expression of TRANCE/RANK L, a critical osteoclast differentiation factor, on mesenchymal cells such as osteoblasts and bone marrow stromal cells **③**. In addition, TRANCE/RANK L is expressed by activated T cells themselves **④**. Binding of osteoblast- or T cell-expressed TRANCE/RANK L to the RANK receptor on osteoclast precursor cells promotes NFATc1-mediated osteoclast differentiation and activation **⑤**, leading to an increase in bone resorption **⑥**. Due to their ability to induce TRANCE/RANK signaling and negatively impact bone homeostasis, activated T cells are now believed to play an integral role in the pathogenesis of rheumatoid arthritis. Whether Th1 or Th17 cells are the primary regulators of this process is still under investigation.

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R&D Systems Tools for Cell Biology Research™

Cytokine-mediated Bone Resorption in Rheumatoid Arthritis

Normal Bone Homeostasis is Maintained by Coupling of Bone Resorption & Bone Formation

