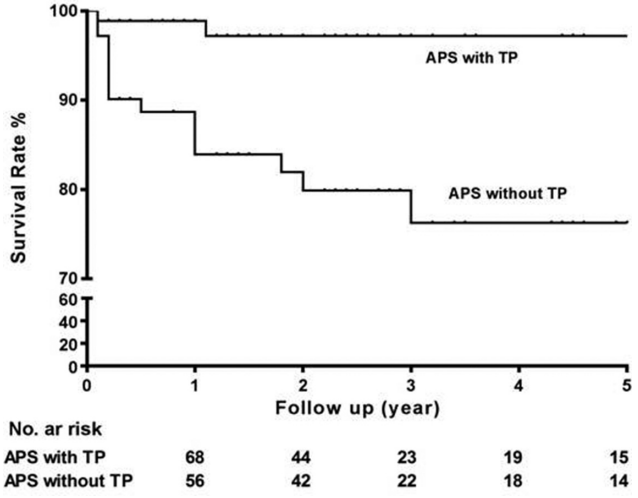
Abstract 69 Table 1 Baseline characteristics of APS patients

Clinical characteristics	Prevalence	Thrombotic event	
		No (N=51)	Yes (N=109)
Female, n/%	110(68.8%)	42(82.4%)	68(62.4%)
Age, year, mean+SD	36.5±14.9	34.2±14.9	37.4±14.9
Thrombotic events			
Arterial thrombosis	59(36.9%)	-	59(54.1%)
Venous thrombosis	72(45.0%)	-	72(66.1%)
Coexist of arterial and venous thrombosis	22(13.8)	-	22(20.2%)
Systemic autoimmune diseases	79(49.4%)	33(64.7%)	46(42.2%)
Thrombophylic risk factors			
Smoking	8(5.0%)	2(3.9%)	5(5.5%)
Dyslipidemia	20(12.5%)	6(11.8%)	14(12.8%)
HTN (systolic>140)	24(15.0%)	7(13.7%)	17(15.6%)
ACL	88(55.0%)	32(62.7%)	56(51.4%)
β2GP1	79(49.4%)	31(60.8%)	48(44.0%)
Lupus anticoagulants	114(71.3%)	39(76.5%)	75(68.8%)
Tri-positive	41(25.6%)	21(41.2%)	20(18.3%)
Thrombocytopenia	71(44.4%)	23(45.1%)	48(44.0%)
Hypocomplementaemia	59(36.9%)	25(49.0%)	34(31.2%)



Abstract 69 Figure 1

differences were found in the clinical presentation of the APS according to the presence or absence of any of these antibodies. During the 10 year period, 16 (10.0%) patients (8 female and 8 male) died. The overall 1, 3, and 5 year survival rate was 92.6%, 89.1% and 87.1%, respectively. The most common causes of death were severe thrombotic events, including pulmonary embolism, strokes and myocardial infarction (43.8% of total deaths), infections (18.8%). COX proportional hazard model show thrombocytopenia is the independent prognostic factor of mortality (HR 8.228, 95% CI 1.866–36.282).

Conclusions Patients with APS develop significant morbidity and mortality despite current treatment. More attention should be devoted to APS patients with thrombocytopenia.

New therapies and therapeutic targets – other autoimmune diseases

70 5-AMINOLEVULINIC ACID COMBINED WITH FERROUS IRON AMELIORATE GRAFT-VERSUS-HOST-INDUCED SYSTEMIC SCLEROSIS IN THE MOUSE

[1]M Fujino*, [2]C Liu, [3]X Yang, [4]P Zhu, [2]S Li, [5]H Ito, [5]K Takahashi, [5]T Tanaka, [5]M Nakajima, [4]J Zhuang, [3]H Zou, XK Li[2]. [1]*National Institute of Infectious Diseases, National Institute of Infectious Diseases, Tokyo, Japan*; [2]*National Research Institute for Child Health and Development, Division of Transplantation Immunology, Tokyo, Japan*; [3]*Fudan University, Institute of Rheumatology- Immunology and Allergy, Shanghai, China*; [4]*Guangdong General Hospital- Guangdong Academy of Medical Sciences, Guangdong Cardiovascular Institute, Guangzhou, China*; [5]*SBI Pharmaceuticals Co.- Ltd, SBI Pharmaceuticals Co.- Ltd, Tokyo, Japan*

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Background and aims Scleroderma or systemic sclerosis (SSc) is a clinically heterogeneous rheumatologic autoimmune disease characterised by skin, internal organs and blood vessels, and there is no effective therapy. The purposes of current study are to develop a model of GvHD-induced scleroderma that more fully represents human condition, and to investigate the effects of 5-aminolevulinic acid (5-ALA), an intermediate of heme synthesis, enhance HO-1 activity to cleave heme to form biliverdin, CO, and iron on this model.
Methods Scl-GvHD was induced by injection of lymphocytes from B10.D2 mice into BALB/c mice deficient in mature T and B cells (recombination activating gene 2 null mice).
Results We successfully established an scl-GvHD model, which is similar to the human disease particularly in the skin, progressive inflammation and fibrosis of internal organs including lung, kidney, and liver. We found that treatment with 5-ALA and iron (Fe^{2+}) significantly reduced progressive inflammation and fibrosis in the skin and ear. Furthermore, by quantitative real-time PCR analysis, 5-ALA and Fe^{2+} suppressed the inflammatory cytokines and TGF-β, type I collagen mRNAs expression. These results indicate that combination treatment with 5-ALA and Fe^{2+} exhibited a protective effect on tissue fibrosis and inflammation of scl-GvHD model mice.
Conclusions The model of GvHD-induced SSc has shown most of symptoms of human disease and is likely to contribute to better understanding of the disease mechanism. Furthermore, efficacy of the 5-ALA has important implication for clarifying the mechanism of HO-1 activity in autoimmune diseases, and may provide a favourable opportunity for clinical therapy.

71 ACTIVATION OF MGLUR7 ATTENUATES THE DEVELOPMENT OF ALLERGY-INDUCED ANAPHYLAXIS

[1]T Gao*. [1]*Uppsala Universitet, Neuroscience, Uppsala, Sweden*

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Background and aims Allergy is a common condition that is caused by an overreaction of the immune system to foreign substances. Severe allergic reactions can result in a systemic life-threatening state referred to as an anaphylactic shock. The progression of the anaphylactic reaction is hard to control after onset, and there is no effective prophylactic treatment available. Recently, mice deficient of the group III metabotropic glutamate receptor mGluR7 were shown to display an anaphylactic-like behaviour when exposed to peripheral histamine, suggesting that mGluR7 works as a neuronal brake on peripheral neurons involved in allergy and anaphylaxis. However, the role of mGluR7 in allergen-induced anaphylaxis is still unknown.
Methods In the PCA model, on the first day, BALB/C mice were lightly anaesthetised with isoflurane and their left ears were intradermally (i.d.) injected with a monoclonal antibody (IgE directed against OVA- trinitrophenol (TNP), 1 µg in 10 µl PBS), whereas the right ears were used as controls (receives an i.d. injection of 10 µl PBS as vehicle). The PCA reaction was induced 24 hours later by an intravenous injection of 50 µg OVA-TNP in 200 µl of 2% Evans blue in PBS.
Results Here, we show that central activation of mGluR7 dampens the development of allergen-induced anaphylaxis as intrathecal, but not intraperitoneal, prophylactic administration of the mGluR7 allosteric agonist N, N'-dibenzhydrylethane-1, 2-diamine dihydrochloride [ML1] AMN082 attenuated the development of passive cutaneous anaphylaxis in mice.
Conclusions Activating the mGluR7 system thus represents a potential preventive treatment for anaphylaxis.

72 IMPLICATIONS OF AUTOPHAGY FOR FUNCTINAL CHANGES OF RHEUMATOID ARTHRITIS FIBROBLAST-LIKE SYNOVIOCYTES

[1]JM Kim*, [2]J Bang, [3]YG Jeong, [4]CH Lee, [1]CN Son, [1]SH Kim. *[1]Keimyung University Dongsan Medical Centre, Division of Rheumatology- Department of Internal Medicine, Daegu, Republic of Korea; [2]Keimyung University Graduate School, Medicine, Daegu, Republic of Korea; [3]Changwon Fatima Hospital, Division of Rheumatology- Department of Internal Medicine, Changwon, Republic of Korea; [4]School of Medicine- Wonkwang University, Division of Rheumatology- Department of Internal Medicine, Iksan, Republic of Korea*

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Background and aims Rheumatoid arthritis (RA) is characterised by exaggerated synovial proliferation in which interleukin-17A (IL-17A) plays a key role. Recently several evidences support the implication of autophagy in the pathogenesis of RA. The aims of this study are (1) to evaluate whether IL-17A influences on autophagic flux in RA synovium and (2) to investigate whether the modulation of autophagy can regulate migration and proliferation of fibroblast-like synoviocytes (FLS) from the patients with RA (RA-FLS) under inflammatory milieu.
Methods FLS from the patients with RA or osteoarthritis (OA) were cultured with IL-17A and/or autophagy regulators. The expression of marker proteins for autophagic flux or the formation of autophagolysosome was analysed by western blot or immunofluorescence study. A migration scratch assay was used to assess FLS migration. Proliferation of FLS was determined by the viable cell count using trypan blue.
Results LC3 conversion from LC3-I to LC3-II was increased in RA-FLS than in OA-FLS. IL-17A upregulated the expression of LC3B, Atg5, Beclin1, LAMP1 in RA-FLS. The accumulation of p62 was also prominent in RA-FLS. Migration and proliferation of FLS stimulated by IL-17A was suppressed by Bafilomycin A1 which prevented the formation of autophagolysosomes. P62-silencing enhanced IL-17A-induced autophagy activation in RA-FLS.
Conclusions This study reveals that IL-17A stimulates autophagy and that intervention of autophagy can control IL-17A-induced migration and proliferation of FLS. Our results also provide additional evidence for a significant role of autophagy in the pathogenesis of RA. Thus, we suggest that autophagy might be a potential therapeutic target for the management of RA.

73 ANTIARTHRITIC EFFECT OF CROCETIN AGAINST ADJUVANT INDUCED AUTOINNUME DISEASE VIA SUPPRESSION THE NF-KB EXPRESSION AND ACTIVATING OF HEM OXYGENASE (HO)-1/NUCLEAR FACTOR-E2-RELATED FACTOR SIGNALLING PATHWAY

[1]V Kumar*, [2]P Bhatt. *[1]Sam Higginbottom Institute of Agriculture – Technology and Sciences, Pharmaceutical Sciences, Allahabad, India; [2]Jamia Hamdard, Pharmacy, New Delhi, India*

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Background and aims Rheumatoid arthritis (RA) is chronic autoimmune diseases, which inducing the cartilage obliteration, synovial joints destruction and typically producing the symmetrical inflammation, which further leads to disability, demolition and deformity into the joint. The aim of the current study was to scrutinise the anti-arthritic potential of crocetin in formaldehyde induced inflammation and complete freund's adjuvant (CFA) induced arthritis.
Methods Formaldehyde used for the induction of acute inflammation and CFA used for induction the arthritis. Both method, the rats were divided into different groups and each group contains the 6 rats. The different doses of crocetin (10, 20 and 40 mg/kg) was used in this model. The body weight, arthritic index were scrutinised at regular interval. Hepatic and antioxidant parameter were determined, respectively.
Results Crocetin dose dependently reduced joint inflammation as support via reduce the joint diameter and decreased inflammatory cell infiltration. Crocetin showed the improvement the synovium redox status (down-regulation in MDA and GSH and boost the CAT and SOD level). Crocetin significantly reduced the expression of inflammatory marker viz., TNF-α. Crocetin enhanced The HO-1/Nrf-2 and reduced the NF-κB mRNA expression in adjuvant joint. Additionally, crocetin treatment decreased the expression of degrading enzymes such AS MMP-3 and MMP-9 in adjuvant induced arthritic rats.
Conclusions Collectively, we can conclude that crocetin showed the anti-arthritic effect via down-regulating the NF-κB and Nrf-2/HO-1 pathway.

74 INFLAMMATORY Vδ2 T CELLS CHEMOTAXIS TO THE JOINTS AND CONTRIBUTE TO THE PATHOGENESIS OF RHEUMATOID ARTHRITIS

W Mo*, S Yin, H Chen, X Zhang. *Beijing peking union medical college hospital, rheumatology, Beijing, China*

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