regulated and involves numerous factors, including calcium deposal and other bone remodelling factors.²

Objectives: The objective of this study was to find a signature of miRs linked both to osteoporotic fracture risk and abdominal aortic calcification (AAC).

The first outcome was the link between miRs levels at baseline and incident osteoporotic fractures (IOF) during 20 years; the second outcome was the link between miRs levels at baseline and the increase in AAC during 17 years.

Methods: Post-menopausal women older than 50 years from the OFELY cohort (Os des Femmes de LYon) were selected if they had available serums at inclusion, and available data for each outcome.

3 miRs selected after literature review because of their impact on vascular calcification and bone turnover (miRs 26a-5p, 34a-5p, and 223-5 p) were measured at baseline. Bioassays of miRs was conducted with miRCURY Biofluids (Exiqon) extraction kit, TaqMan Life Technologies protocol, and QuantStudio 7 flex (Applied Biosystems) for RNA quantification. Results are expressed by relative quantification of Cycle threshold (Ct).

Results: A sample of 434 age-matched women (63 [57–72] years old), 50% with incident osteoporotic fracture during the 20 years of follow-up, was included. 183 women had available data to explore AAC; 93 had an increase in Kauppila score in 17 years (58 [55–61] years old), 90 did not (55 [53–58] years old).

No significant link was underlined between miRs and IOF (miR-26: 1.06 [0.85–1.27] vs 0.99 [0.85–1.17], $p=0.07$; miR-34: 1.15 [0.53–1.87] vs 1.26 [0.60–2.07], $p=0.35$; miR-223: 1.01 [0.68–1.43] vs 1.05 [0.72–1.56], $p=0.32$).

No miR was significantly linked to an increase in AAC (miR-26: 1.09 [0.94–1.28] vs 1.10 [0.89–1.30], $p=0.95$; miR-34: 0.78 [0.46–1.21] vs 0.73 [0.38–1.50], $p=0.90$; miR-223: 0.97 [0.69–1.22] vs 0.78 [0.56–1.22], $p=0.11$).

Conclusions: No association was observed between the 3 tested miRs and IOF or increase in AAC. Larger studies are necessary to select interesting epigenetic pathways reproducible on wider population.

REFERENCES:

- [1] Krzeszinska JY, Wei W, Huynh H, Jin Z, Wang X, Chang T-C, et al. miR-34a Blocks Osteoporosis and Bone Metastasis by Inhibiting Osteoclastogenesis and Tgfr2. *Nature* 2014 Aug 28;512(7515):431.
- [2] Szulc P. Abdominal aortic calcification: A reappraisal of epidemiological and pathophysiological data. *Bone* 2016 Mar;84:25–37.

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AB0009 GENETIC ASSOCIATION OF MITOCHONDRIAL DNA POLYMORPHISMS WITH BEHÇET'S DISEASE IN A KOREAN POPULATION

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Background: Behcet's disease (BD) is an inflammatory multi-genetic disorder with unknown etiology. In the previous study, we sequenced whole mitochondrial nucleotides from blood of 20 BD patients and 10 sex-, age-matched healthy controls, m.248A>G, m.709G>A, m.3970C>T, m.6392T>C, m.6962G>A, m.10310G>A, m.10609T>C, m.12406G>A, m.12882C>T, m.13928G>C, m.16129G>A, and m.16304T>C were more frequently observed in patients group than healthy control without statistical significance. While, m.304C>A, m.3010G>A, m.4883C>T, m.5178C>A, and m.14668C>T were more frequent in control group ($p=0.008$, 0.026, 0.007, 0.007, and 0.026, respectively). m.16182A>C, m.16183A>C, m.16189T>C were associated with uveitis ($p=0.041$, 0.022, and 0.014, respectively).

Objectives: We performed a follow-up study to validate these possible associations in larger groups.

Methods: Whole blood or buffy coat were collected from 98 BD patients from four university hospitals located in Chung-Cheong district of Republic of Korea, 196 age-, sex-matched healthy controls from Konyang University Hospital. Above mentioned 20 targeted mitochondrial DNA (mtDNA) genomes were analysed using MassARRAY[®] system (Agena Bioscience, Inc., San Diego, CA, USA) for m.709, m.3010, m.4883, m.6392, m.6962, m.10310, m.10609, m.12406, m.12882, m.13928, and m.14668; Sanger sequencing for m.248, m.304, m.5178, m.16129, m.16182, m.16183, m.16189, and m.16304; and TaqMan-based genotyping assay (Applied Biosystems, Foster City, CA, USA) for m.3970. Results were compared with the revised Cambridge Reference Sequence (rCRS). Chi square or Fisher's exact test were used to analyse association of mtDNA

alterations between groups and between mtDNA alterations and clinical/laboratory characteristics.

Results: Presence of m.248A>G, m.304C>A, m.709G>A, m.3010G>A, m.3970C>T, m.4883C>T, m.5178C>A, m.6392T>C, m.6962G>A, m.10310G>A, m.10609T>C, m.12406G>A, m.12882C>T, m.13928G>C, m.14668C>T, m.16129G>A, and m.16304T>C were not differentiated by the groups. However, m.16812A>C was more frequently observed in the patient group than control [22 (22.4%) vs. 24 (12.2%), $p=0.061$], and it was significantly associated with HLA-B51 positivity ($p=0.011$), arthralgia ($p=0.043$) and methotrexate use ($p=0.02$), and was not associated with uveitis in the follow-up study. Among clinical and laboratory characteristics in BD patients, thrombosis was more frequently observed in male patients than female patients [7 (22.6%) vs. 0 (0%), $p<0.001$], and WBC counts were lower in female patients [3642.3±3413.6 vs. 5200.8±3912.7, $p=0.048$].

Abstract AB0009 – Table 1

RCRS position	Alteration	BD (n=98)	HC (n=196)	p-value	OR* (95% CI)
248	A del	12 (12.2%)	23 (11.7%)	1.000	1.050 (0.499~2.209)
304	C>A	0 (0%)	0 (0%)	NA	NA
709	G>A	24 (24.5%)	35 (17.9%)	0.236	1.492 (0.929~2.686)
3010	G>A	19 (19.4%)	50 (25.5%)	0.307	0.702 (0.387~1.273)
3970	T>C	14 (14.3%)	19 (9.7%)	0.327	1.553 (0.743~3.246)
4883	C>T	25 (25.5%)	59 (30.1%)	0.494	0.795 (0.460~1.374)
5178	C>A	25 (25.5%)	59 (30.1%)	0.494	0.795 (0.460~1.374)
6392	T>C	14 (14.3%)	19 (9.7%)	0.327	1.553 (0.743~3.246)
6962	G>A	12 (12.2%)	18 (9.2%)	0.540	1.380 (0.636~2.993)
10310	G>A	16 (16.3%)	24 (12.2%)	0.434	1.398 (0.705~2.774)
10609	T>C	12 (12.2%)	16 (8.2%)	0.361	1.570 (0.711~3.463)
12406	G>A	12 (12.2%)	17 (8.7%)	0.447	1.469 (0.672~3.213)
12882	C>T	12 (12.2%)	16 (8.2%)	0.361	1.570 (0.711~3.463)
13928	G>C	14 (14.3%)	19 (9.7%)	0.327	1.553 (0.743~3.246)
14668	C>T	19 (19.4%)	49 (25.0%)	0.353	0.722 (0.398~1.310)
16129	G>A	23 (23.5%)	42 (21.4%)	0.804	1.124 (0.631~2.005)
16182	A>C	22 (22.4%)	24 (12.2%)	0.061	2.062 (1.089~3.906)
16183	A>C	32 (32.7%)	42 (21.4%)	0.092	1.766 (1.026~3.040)
16189	T>C	42 (42.9%)	64 (32.7%)	0.112	1.547 (0.939~2.548)
16304	T>C	15 (15.3%)	18 (9.2%)	0.170	1.787 (0.859~3.720)

Conclusions: We performed a follow-up study to validate possible associations between BD and 20 mtDNA alterations. m.16812A>C could be associated with BD and its several clinical or laboratory characteristics a Korean population.

REFERENCES:

- [1] Xavier JM, Shafiee NM, Ghaderi F, Rosa A, Abdollahi BS, Nadjai A, et al. Association of mitochondrial polymorphism m.709G>A with Behçet's disease. *Ann Rheum Dis* 2011;70:1514–6.
- [2] Kwon MH, Joung CI. Genetic Associations of Mitochondrial DNA Polymorphisms with Behçet's Disease in a Korean Population: A Pilot Study. *J Rheum Dis* 2016;23(1):23–9.

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AB0010 EVALUATION OF SENSITIVITY TO DNA DAMAGING AGENTS AND EFFICIENCY OF DNA REPAIR IN HUMAN PERIPHERAL BLOOD MONONUCLEAR CELLS FROM PATIENTS WITH DERMATOMYOSITIS AND POLYMYOSITIS

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Background: Idiopathic inflammatory myopathies (IIM) being one of the connective tissue diseases are a group of diseases with not fully understood pathology. The common features are production of autoantibodies, abnormal immune response against self-antigens and inflammatory process leading to destruction of muscle cells and internal organ involvement. Patients with inflammatory myopathies have higher risk of developing cancers. One of the processes that can