"Buy one - get 4 free" - RMDs and comorbidities

Keywords: Mental health, Pain, Patient information and education


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Table 1.

<table>
<thead>
<tr>
<th>Themes</th>
<th>Quotes</th>
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</thead>
<tbody>
<tr>
<td>Content and delivery of the programme</td>
<td>“Each of the four sections were all very relevant”</td>
</tr>
<tr>
<td>Understanding the effects of symptoms on their own and combined</td>
<td>“Now I know that I can sleep better when I manage my pain, and I can manage my pain by managing fatigue”</td>
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<tr>
<td>Implementing the knowledge gained from the programme</td>
<td>“I’m in a different place today than I was four weeks ago. I am doing well now. So, for me the goal-setting is excellent”</td>
</tr>
<tr>
<td>Impact of the FAME-W on symptoms and work</td>
<td>“Just a lightbulb moment to say, great, this is not my fault, because you can blame yourself for all the symptoms”</td>
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</table>

Conclusion: Preliminary results show that participants found the online FAME-W to be effective, relevant, reassuring, and helpful. These results suggest that work-related self-management skills are essential in assisting participants with symptom management in the workplace. Furthermore, these preliminary results suggest that the online format of FAME-W may be helpful for individuals with inflammatory arthritis to stay in work and it may become a standard part of clinical care for occupational therapists.

REFERENCES:

OP0011

EPITRANSCRIPTOME EDITING REPRESENTS AN ADDITIONAL LAYER OF IMMUNE-RELATED EPIGENETIC MEMORY OF MONOCYTES IN HUMAN SYSTEMIC LUPUS ERYTHEMATOSUS (SLE)

Keywords: Systemic lupus erythematosus, Genetics/Epigenetics, Innate immunity

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Background: Monocytes are key effector cells in human systemic lupus erythematosus (SLE). Cytokine production, antigen presentation and initiation/amplification of tissue inflammation[1] represent some of output functions of transcriptional rewiring within the inflammatory milieu of the disease. Epitranscriptome includes all functionally relevant changes to the RNA level (transcriptome), reforming RNA metabolism and fine-tuning gene expression[2].

Objectives: To construct the transcriptional map of monocytes in lupus patients and impose the A-to-I base editing events as co-factor of the disease pathogenesis.

Methods: A cohort of 8 SLE patients (SLEDAI(8) and 5 age/sex matched healthy volunteers was used for this study. PBMCs were obtained through Ficoll centrifugation and monocytes were isolated with CD14+ magnetic bead selection. Total RNA was extracted and mRNA libraries were generated. Single-end 75-bp mRNA sequencing was performed on Illumina NextSeq 500. Differential expression analysis was performed using edgeR package. Genes with a false discovery rate of ≤0.05 and a fold change of ≥1.5 were considered statistically significantly deregulated. Significant differentially expressed genes (DEGs) were used for pathway and gene ontology (GO) analysis using gProfiler web-server[3]. For base editing events, JACUS2A was utilized for base calling and list refinement was performed by the following criteria: (a) known editing sites based on REDPortal, (b) the number of reads with “G” at the putative editing site is at least 10 and (b) the editing level per sample, defined as 100 nG/[nA+nG], is at least 20%, (c) JACUS2A call-2 score for differential editing between SLE and healthy was 1.5.

Results: Differential gene expression analysis produced a list of 764 statistically significant genes, 492 of which are upregulated and 272 downregulated. Gene enrichment analysis concluded that DEGs are participating in transcription regulation, signal transduction, regulation of immune processes and apoptosis/cell death. IL-17 signaling pathway and RNA metabolic process are part of the patho-genetic profile of lupus monocytes. Interestingly, ADAR1, key regulator of RNA editing, is upregulated (logFC=0.76, pval=0.018). A-to-I editing events found to be significant, contain among others GDI2 (UTR3 region/signaling mediator), ARPC2 (Alu region/actin polymerization) and CERK (UTR3 region/inflammation) regulatory regions. A complex network of transcriptional and epitranscrip-tomic alterations embeds immune-related genes and affects multiple cellular processes.

Conclusion: Transcriptional machinery and signal transduction molecules exhibit abnormal expression during transcriptional rewiring in monocytes of lupus patients. Our data support RNA editing as an additional player of immune-related epigenetic memory of monocytes, further integrating inflammatory cues and enhancing autoimmune identity of the disease.

REFERENCES:

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How to treat older people with RMD

OP0012-HPR

CENTRAL SENSITIZATION HAS MAJOR IMPACT ON DISEASE ACTIVITY, FUNCTIONAL DISABILITY AND FRAILTY IN PATIENTS WITH RA

Keywords: Patient reported outcomes, Rheumatoid arthritis, Outcome measures

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Background: Central sensitization (CS) assessed with Central sensitization Inventory (CSI) is significantly associated with functional disability and frailty in patients with rheumatoid arthritis (RA). Frailty is common in RA and associated with hospitalization and mortality. Frailty in RA is dynamic in nature, for some, may be ameliorated through controlling disease activity and functional disability.

Objectives: Our aim was to investigate the prevalence of CS in patients with RA and its association with measures of disease activity, functional disability, and frailty.

Methods: We administered to all the subjects in the study the CS inventory (CSI), a questionnaire that has been used for the diagnosis of CS. Demographic and sociodemographic characteristics and measures such as disease activity (DAS28), functional disability (ROAD) and Kihon Checklist (KCL) screening tool to identify community-dwelling adults vulnerable to frailty potentially at risk of becoming dependent. Patients with fibromyalgia were excluded from the study.

Results: Of the 192 included RA patients, mean CSI score was 36.7 ±15.5 and 36.5% scored >40, which indicates a high probability of CS. Mean DAS28 score was 6.8 ± 12.4 and mean RAID 5.0 ± 2.0. A CSI score >40 was significantly associated with higher RAID (mean 5.7 vs. 4.6, F-ratio 12.28; p<0.001), higher

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