effects of pure cocoa on a mechanically-induced headache model in healthy individuals. Serum β-endorphin concentrations were also measured to identify if any changes occurred in response to cocoa consumption.

Methods: Healthy volunteers (8 men and 7 women, average age: 22 ± 1.8 years) participated in a crossover study (approval number: N-20160015) consisting of two sessions with consumption of water or cocoa. A mechanical headband (custom-made, Aalborg University) was utilized to induce a moderate headache with pain rated as 4 on a Visual Analogue Scale (VAS_{0-10}). In each session Pressure Pain Threshold (PPT) was measured by a hand held algometer at temporalis muscles and blood samples were collected to assess β-endorphin levels by ELISA. ANOVA analysis and independent two-sample *t*-tests were performed for comparisons. $P < 0.05$ was considered as significant.

Results: Consumption of cocoa compared to water did not change the pressure sensitivity in the craniofacial region without mechanical headband. No changes occurred in PPT with the mechanical headband either. Regardless of substance consumption (water/cocoa), endorphin levels remained unchanged.

Conclusions: Findings demonstrated that pure cocoa in the applied concentration and within the timeline of this study did not seem to exert any analgesic or pro-algesic effect on the mechanical sensitivity of craniofacial muscles or β-endorphin levels.

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The impact of naloxegol treatment on gastrointestinal transit and colonic volume

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Aims: Opioid treatment is associated with gastrointestinal (GI) side effects, known as opioid-induced bowel dysfunction (OIBD). Symptoms of OIBD are caused by opioid receptor activation in the enteric nervous system, which results in increased GI transit time and increased faecal volume in the colon. OIBD can be experimentally induced in healthy participants through oral oxycodone treatment. The aim of this study was to investigate whether administration of naloxegol, a peripherally restricted opioid antagonist, could reduce GI symptoms, GI transit time, and colorectal volume, using an experimental model of OIBD.

Methods: In a double blind crossover trial, twenty-five healthy males were randomly assigned to a six day treatment of oral oxycodone in combination with either oral naloxegol or placebo. At baseline and at day six, participants filled in the Patient Assessment of Constipation Symptom questionnaire, and colorectal volume was quantified with a magnetic resonance imaging method. Participants swallowed a small electromagnetic capsule, which allowed determination of total and segmental GI transit times, using the 3D-Transit system.

Results: In the established model of oxycodone induced OIBD, fewer GI symptoms were observed during naloxegol treatment, compared to placebo ($P < 0.01$). Naloxegol decreased median total transit time by 27% (56 *vs* 71 h, $P < 0.05$) and decreased colorectal transit time by 33% (45 *vs* 59 h, $P < 0.01$), compared to placebo. No difference in colorectal volume was found between the two treatments.

Conclusions: In an experimental model of OIBD, GI symptoms and GI transit time were reduced during treatment with naloxegol, compared to placebo. However, naloxegol treatment did not reduce colorectal volume. These findings add information on the potential of naloxegol to be used in prevention and treatment of OIBD.

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Preoperative downregulation of long-noncoding RNA Meg3 in serum of patients with chronic postoperative pain after total knee replacement

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Aims: The incidence of chronic pain after total knee replacement (TKR) is approx. 20%, why preoperative risk factors for the development of chronic postoperative pain are highly warranted. Studies have indicated that preoperative inflammatory markers hold prognostic information for the development of chronic postoperative pain. Long-non-coding-RNA (lncRNA) regulates the expression of genes related with e.g. inflammation. The current study aimed to investigate the preoperative influence of lncRNA on the development of chronic postoperative pain following TKR.

Methods: 24 patients, who developed chronic postoperative pain and 12 patients with painfree recovery, were sampled from a larger clinical study. Preoperative serum samples were obtained from all patients and analyzed for potential lncRNA candidates. Briefly, total RNA was extracted from serum using miRNeasy Mini kit. cDNA samples were pre-amplified with RT2lncRNAPreAMP Primer Mix that contained specific set of primers to target genes of Human RT2lncRNA Inflammatory Response & Autoimmunity PCR Array. Further, the reaction (25 μl) will be aliquoted into the wells of RT2lncRNA PCR Array Human Inflammatory Response & Autoimmunity. LncRNA-expressions were compared between the two groups using student's *t*-test.

Results: 19 patients were excluded due to the "cut-off" of the statistical analysis's software that not included the samples because of genomic contamination, retro transcription and amplification's low efficiency. A total of 13 patients were included (7 with pain, 6 without pain). The preliminary analysis found that the MEG3-lncRNA (implicated in tumor vascularization suppressing) were downregulated in patients who developed chronic postoperative pain compared to patients with a normal recovery (fold change: −9.56, $P < 0.05$).

Conclusions: Preoperative MEG-3 is downregulated in patients in risk of developing chronic postoperative pain following TKR. Future research, in larger cohorts should further investigate the role of MEG3 and hence the improvement of the cartilage