

## **Distinct cerebral metabolic patterns related to high pain sensitivity in episodic or chronic migraine patients and healthy volunteers**

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**Introduction:** Allodynia, i.e. pain evoked by a non-painful stimulus, is prevalent in chronic pain and in migraine where it augments with disease severity and chronicity [1]. Central sensitization is thought to be the culprit [2]. It is not known, however, which central areas are involved. The aim of the present study was to evaluate whether brain metabolism in subjects that are more sensitive to pain is different between migraine patients and healthy controls.

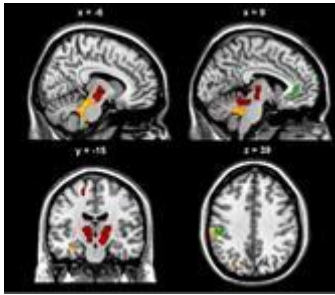
**Subjects and methods:** Quantitative heat sensory testing on the forehead and 18FDG-PET were performed in 55 subjects: 20 healthy volunteers (HV, 21-59 years, 5M), 21 patients with episodic migraine in the interictal phase (MO, age range: 20-63 years, 5M) and 14 patients with chronic migraine (CM, age range: 22-62 years, 1M). The 3 cohorts were subdivided according to the median heat pain threshold into subgroups with low and high pain thresholds. PET results were compared between these subgroups in each cohort. Data analyses were restricted to areas of the pain/salience matrix.

**Results:** There was no significant difference in heat pain thresholds between HV (median: 43.7 °C), MO median: 44.2°C) and CM (median: 43.3°C) ( $p=0.64$ ). In an SPM-ANOVA, a contrast modelling the potential gradual effect of increased differences in pain sensitivity in relation to disease severity showed significant metabolic changes in bilateral thalamus and midbrain ( $p < 0.001$ ). Additional analyses revealed that hypometabolic areas in subgroups with a low heat pain threshold differed between HV (anterior cingulate and somatosensory cortices), MO (lower pons and somatosensory cortex) and CM (midbrain and thalamus) (Figure 1).

**Conclusion:** Overall migraine patients do not have reduced heat pain thresholds. However, hypometabolic areas related to high thermal pain sensitivity are strictly cortical in HV, but comprise the pons in episodic migraine and are restricted to midbrain and thalamus in chronic migraine. The distinct central correlates of heat pain sensitivity in migraine patients might therefore represent a biomarker of migraine and its chronification. Legend to figure Figure 1. Hypometabolic areas in low pain threshold subgroups in HV (green), MO (orange) and CM (red).  $p < 0.01$  for display purpose.

**Résumé en Français:** Titre: Métabolisme cérébral distinct en relation avec la sensibilité à la douleur entre sujets sains, migraine épisodique et migraine chronique. Les migraineux ont une sensibilité anormale à la douleur. Les mécanismes cérébraux en sont inconnus. Nous avons comparé le métabolisme cérébral chez des sujets sains et chez des migraineux épisodiques ou chroniques et corrélié les résultats avec le seuil douloureux. Les aires cérébrales hypométaboliques liées à un seuil douloureux bas diffèrent entre groupes: régions corticales chez les sujets sains, aires corticales et sous-corticales dans la migraine épisodique, régions sous-corticales dans la migraine chronique. Le contrôle central de la douleur semble modifié distinctement dans les formes de migraine, ce qui pourrait en constituer un biomarqueur et avoir des implications thérapeutiques.

**Figure 1**



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### **References**

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