Axon-reflex cutaneous vasodilatation is impaired in type 2 diabetic patients receiving chronic low-dose aspirin

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Résumé en anglais	Low-dose aspirin is largely but non-homogeneously used in primary prevention of cardiovascular complication in type-2 diabetic patients. We hypothesised that low-dose aspirin could interfere with the cutaneous neurovascular responses in type-2 diabetic patients. Galvanic current-induced vasodilatation (CIV) is an original non-noxious integrative model of neurovascular interaction and is impaired under low-dose aspirin in healthy subjects. Twenty type-2 diabetic patients (ten not receiving aspirin: D-NA and ten regularly receiving ≤ 150 mg/day aspirin: D-A), and ten age-, BMI-, and gender-matched non-diabetic control volunteers (MC), underwent macro- and microvascular investigations, including: CIV, acetylcholine (ACh) and sodium nitroprusside (SNP) iontophoresis, post-occlusive hyperemia (POH), neuropathy symptom (NSS) and disability (NDS) scores, and thermal and vibration sensory thresholds. Results are presented as median [25–75 centile] and microvascular results are expressed in multiple from baseline conductance (%Cb). CIV was 554 [349–769] %Cb in MC, 251 [190–355] %Cb in D-NA and 159 [136–202] %Cb in D-A ($p < 0.05$). No differences were observed between the three groups except for CIV, which is impaired in diabetic patients and further impaired in those regularly receiving low-dose aspirin, while other macrovascular, microvascular and clinical-sensitivity investigations show no significant difference. Potential clinical markers for the impairment of the neurovascular interaction are still required in diabetes. Correlation of the CIV response with the risk of cutaneous complications in diabetic patients remains to be tested.
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