Correspondence

Parchment-skin illusion: sound-biased touch

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Our brains continuously bind information obtained through many sensory channels to form solid percepts of objects and events. Usually these pieces of information complement and confirm each other, thereby improving the reliability of our perception [1]. But incongruities between the sensory inputs may result in unexpected percepts due to intersensory interactions, as in the well-known audiovisual McGurk illusion [2].

Audiotactile interactions have remained largely unexplored [1], although Paul von Schiller [3] noted in 1932 that sounds — noise bursts or tones repeated at regular intervals — may affect tactile perception of roughness. We describe here a novel audiotactile interaction, 'parchment-skin illusion', which demonstrates that sounds that are exactly synchronous with hand-rubbing may strongly modify the resulting tactile sensations.

The subjects were seated with forearms supported on their thighs. A microphone close to the hands was recording the sounds produced when the subjects rubbed their palms together in a back-and-forth motion at 1–2 cycles per second. The sounds were played back to the subject through headphones. This audio feedback was either identical to the original sound or modified so that the high frequencies (above 2 kHz) were either dampened by or accentuated by 15 decibels (dB). In addition, the maximum sound intensity, which was adjusted to a comfortable listening level, was attenuated by either 20 or 40 dB, resulting in a randomized experiment of 3×3 block design, as shown in Table 1.

During the pilot sessions several subjects spontaneously reported that the enhanced high-frequency feedback made the palmar skin feel drier, almost resembling parchment paper; this effect was found in 13 out of 17 healthy adults tested. Moisture on the palmar skin typically prevented the phenomenon.

Eleven subjects (six males, five females; age range 25–49 years) who reported the phenomenon in a consistent manner were asked to quantify the tactile sensations on their palms during varying audio feedback conditions on a scale of 0 to 10, referring to a range rough/moist–smooth/dry, respectively. The audio feedback had a very clear effect on the tactile sensation as is evident from Table 1.

When either the proportion of the high frequencies or the average sound level of the auditory feedback increased, the skin started to feel more paper-like, that is, the perceived roughness/moisture of the palmar skin decreased and the smoothness/dryness increased. The effects of both the high-frequency content and the average intensity of the feedback were statistically highly significant (by analysis of variance). Tactile sensitivity — tested with von Frey hairs in two subjects while they performed the task with audio feedback — was not modified during the illusion, as compared with the no-feedback condition with the hands at rest.

An additional experiment with two experienced subjects showed that a delay of the audio feedback by more than 100 milliseconds clearly diminished the illusion. Efficient binding of multisensory inputs evidently requires accurate temporal coincidence, or a temporal window for multisensory integration (as discussed in [4]), which naturally happens when the subjects hear the sounds produced by their own hand movements.

We hypothesize that the parchment-skin illusion reflects an omnipresent intersensory integration phenomenon, which helps the subject to make accurate tactile decisions about the roughness and stiffness of different textures they manipulate.

Table 1

Tactile sensation of skin roughness

Attenuation level	High-frequency feedback		
	–15 dB	**Normal**	**+15 dB**
–40 dB	0.1 ± 0.5	0.7 ± 0.2	2.4 ± 0.4
–20 dB	1.4 ± 0.5	3.8 ± 0.8	7.1 ± 0.6
0 dB	4.5 ± 0.8	6.8 ± 0.6	9.5 ± 0.3

Tactile sensation of relative skin roughness as a function of quality of the auditory feedback; mean ± SEMs of 11 subjects. The original estimates of skin roughness were given on a 0 (rough or moist) to 10 (smooth or dry) scale but as the individual ranges varied from 3.5 to 10, the values were normalized according to the range of each individual before averaging. The individual values were means of two repeated tests.

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MADD is highly homologous to a Rab3 guanine-nucleotide exchange protein (Rab3-GEP)

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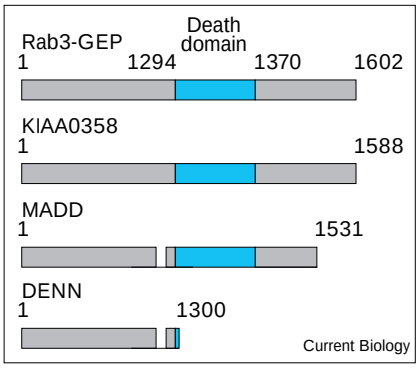
The MADD protein (death domain MAP kinase activator) has recently been identified as a death-domain-containing protein involved in the activation of mitogen-activated protein (MAP) kinase and arachadonic acid release mediated by the tumor necrosis factor receptor type 1 (TNFR1) [1]. The death domain of MADD (amino acids 1281–1356) can bind to the death domain of TNFR1 and can activate MAP kinases. Overexpression of MADD has no effect on the induction of apoptosis, however. Thus, although the presence of a death domain is suggestive of a potential protein–protein interaction, it does not always implicate a protein in the apoptotic process. Sequence analysis of the death domain in the MADD protein was performed using the BLAST local alignment program [2] to search the non-redundant sequence GenBank database at NCBI. Two proteins were identified that had almost identical amino-acid sequences to that of the death domain of MADD (Figure 1). Analysis of the full-length protein sequences of these two proteins using MacVector (Eastman Kodak) and Geneworks (Intelligenetics) protein alignment programs revealed that one of them was the GDP/GTP exchange protein Rab3-GEP [3]. Rab3-GEP has greater than 94% overall identity to MADD. The only significant difference between MADD and Rab3-GEP is in a region amino terminal to their death domains, where 21 amino acids have been deleted in MADD. The other protein with an almost identical death domain to that of MADD was a brain protein of unknown function, KIAA0358. Furthermore, full-length MADD and Rab3-GEP are 87% identical to KIAA0358, although KIAA0358 has a carboxy-terminal 61 amino-acid deletion.

Sequence analysis of the amino-terminal region of the MADD protein identified a fourth protein, DENN, which is highly homologous to MADD (98% identity), Rab3-GEP and KIAA0358, although it lacks the carboxy-terminal 300 amino acids of MADD, including the death domain. DENN was previously identified as a differentially expressed protein in neoplastic tissues [4] and shown to have homology to Rab3-GEP [3] and the product of the *Caenorhabditis elegans aex-3* gene [5]. The *aex-3* gene product has 33% homology to Rab3-GEP, and mutation of *aex-3* results in mislocalization of Rab3 [5].

Rab family members are modified by geranyl–geranyl moieties, cycle between the GDP-bound inactive and GTP-bound active state [3], and have been shown to function in vesicular traffic [3,6]. Other small GTPases, such as Ras, Rac and Cdc42 [7–9], have been shown to play important roles in positive and negative regulation of cell signaling and growth. The identification of MADD as a regulator of TNFR1 function (through its death domain), and the similarity between MADD and Rab3-GEP provides a connection between TNFR1 activation and downstream kinase activity through a guanine-nucleotide exchange protein. The variations that exist among the four identified proteins may have arisen as a result of alternative splicing, thereby generating different isoforms of death-domain-containing proteins. Future studies may determine whether these MADD/Rab3-GEP isoforms are capable of activating specific MAP kinases via GDP/GTP exchange and of effecting TNF-mediated signaling, arachadonic acid release and/or vesicular traffic.

Figure 1



MADD-related isoforms. Blue boxes identify the position of the death domain present in Rab3-GEP (GenBank Accession number U72995), KIAA0358 (AB002356), MADD (U77352) and DENN (U44953).

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