**Limitations of the study**

Some potential limitations are associated with the present study. First, a simple paracentric inversion cannot cause the observed in-frame fusion of *NUP160* and *SLC43A3*. Based on the orientation of the genes and the appearance of chromosome 11s, a simple interstitial deletion is also unlikely. For example, interchromosomal or interchromatid rearrangement may be the cause of the *NUP160-SLC43A3* fusion (1). This point should be clarified in the future.

Angiosarcoma may have other *NUP160*-*SLC43A3* fusion genes with different breakpoints. We performed RT-PCR using angiosarcoma tissue RNAs and primers designed for the other exons of *NUP160* or *SLC43A3*, but did not obtain specific amplification with any of these (data not shown). In addition, we tested the 13 other candidate fusion genes (Supplementary Table4); however they were not detected in tissue RNAs by RT-PCR (data not shown). The presence of the fusion genes in angiosarcomas of other tissues including liver or breast also needs to be determined.

HDMEC originated from a female, while ISO-HAS originated from a male patient (see the karyotypes in Fig.1D). Commercially available HDMECs are generated from either male foreskin or from adult females. We considered that the latter would be more suitable as the control cells because angiosarcoma is usually seen in elderly people, and because the fusion gene is found in both male and female patients.

Furthermore, we injected HDMECs transfected with the fusion gene subcutaneously into nude mice, but we did not observe any tumor formation (data not shown), probably due to the differences between humans and mice. Methods for the development of mouse dermal microvascular endothelial cells have been described (2). However, examining this gene fusion in mouse cells will not provide meaningful information that is relevant to the gene fusion seen in human endothelial cells, because there are distinct differences between mice and humans in the biological behavior and/or evolution of hemangiomas and angiosarcomas as described previously (3,4). An *in vivo* mouse angiosarcoma model using HDMECs transfected with the fusion gene needs to be developed by a future study.

**References**

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