We have extensive evidence that the presence of α-synuclein in transplants varies depending on transplantation methods. We have stained tissue that we obtained from the authors of ref. 3 that yielded results identical to those they originally reported, whereas the results from our transplant series[2] are very different: out of several hundreds of thousands of neurons that we have studied, we have found only one Lewy body–like structure. We have also explored hypotheses[4,5] for why methodological differences could account for a different rate of insoluble α-synuclein deposits in transplants. Our observations indicate that the presence of reactive microglia in a transplant can occasionally correlate with the presence of Lewy body–like structures.

In conclusion, although there clearly can be α-synuclein deposits in tissue transplanted to patients with Parkinson's disease, their presence seems to depend on methodological differences. In addition, the presence of such deposits in our transplant series is so low—even after more than a decade—that it would be hard to imagine that it has any clinical relevance[4].

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Kordower, Halliday and Obeso reply:
We appreciate the opportunity to clarify a salient point in our recent review[1]. Drs. Isacson and Mendez are correct that the percentage of Lewy bodies in grafted neurons (5–8%) that we cited referred specifically to the findings of Kordower *et al.*[2] and that it was not meant to refer to all of the papers referenced in the previous sentence. We are glad to clarify this point. It is interesting to note that Li *et al.*[3] recently reported that 2% and 5% of grafted neurons contained Lewy bodies 12 and 15 years, respectively, after grafting, even though it is true that the cases reported by Mendez *et al.*[4] contained far fewer Lewy bodies.

Hypotheses regarding why Lewy bodies form in long-term transplants have created great interest in the Parkinson's disease community, although nothing has yet been proven. Inflammation and oxidative stress are hypothesized to cause Parkinson's disease itself, and we would therefore not be surprised if these mechanisms have a role in the formation of Lewy bodies, although extensive microglial inflammation occurs in long-term striatal grafts in Huntington's disease patients without the formation of Lewy bodies[5]. We pointed out in our review that the significance of Lewy bodies in terms of cell death or protection is still uncertain. Accordingly, we agree with our colleagues that, given that Lewy bodies are relatively few in number, their contribution to and impact in grafted neurons may not be determinant factors for the success of cell replacement therapy, as pointed out elsewhere[6]. At the same time, the fact that some young transplanted cells do show the pathological hallmark of Parkinson's disease may signal a potential limitation of cell replacement and similar approaches[7].

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Sodium guidelines should be taken with a grain of salt

To the Editor:
Stephen Strauss's news feature[1] on low-sodium alternatives to table salt is predicated on the notion that salt consumption is currently at unhealthy levels. But the clinical evidence supporting salt intake reduction to the most commonly recommended level of 1.5–2.3 grams of sodium per day is almost nonexistent. The only basis for these two figures that I have come across is in the Dietary Reference Intakes introduced by the US Institute of Medicine (IOM) to inform healthcare and nutritional guidelines, yet the same policy documents also state the average required level of sodium "could not be established because of inadequate data from dose-response studies"[2].

The IOM arbitrarily set the adequate intake instead of stating the need to collect more clinical data. This opinion was based neither on the amount needed to replace the sodium lost in a day (about half a gram per day) nor on the human physiological sodium control mechanism, the renin-angiotensin-aldosterone system, which is stimulated at anything below three grams of sodium per day to signal the kidneys to begin recouping the element.

The upper sodium limit of 2.3 grams was, again, subjective. In deference to Avogadro, the level was set at exactly 100 millimoles, on the notion that it would do no harm. In a short period of time, the Dietary Reference Intake of salt morphed into urban legend and is now regarded as established fact, particularly by the media. As a result, the demand for more reliable evidence has been supplanted by a demand for action on population-wide salt reduction, regardless of all evidence that no such action is warranted. For instance,