The sound of many hands clapping

Tumultuous applause can transform itself into waves of synchronized clapping.

An audience expresses appreciation for a good performance by the strength and nature of its applause. The thunder of applause at the start often turns quite suddenly into synchronized clapping, and this synchronization can disappear and reappear several times during the applause. The phenomenon is a delightful expression of social self-organization that provides an example on a human scale of the synchronization processes that occur in numerous natural systems, ranging from flashing Asian fireflies to oscillating chemical reactions. Here we explain the dynamics of this rhythmic applause.

We investigated the mechanism and development of synchronized clapping by making a series of measurements focusing on the collective aspects of the self-organization process as well as on the behaviour of the individuals in the audience. We recorded several theatre and opera performances in Romania and Hungary by using a microphone placed on the ceiling of the hall (Fig. 1a). Typically, after a few seconds of incoherent random clapping, a periodic signal developed (a sign of synchronized clapping), visible in Fig. 1a as pronounced spikes. The transition is also captured by the order parameter (Fig. 1c), which increases as the periodic signal develops, and decreases as it disappears.

Although synchronization increases the strength of the signal at the moment of the clapping, it leads to a decrease in the average noise intensity in the room (Fig. 1d). This is surprising, as the driving force for synchronization would be expected to reflect the desire of the audience to express its enthusiasm by increasing the average noise intensity.

The origin of this conflict between the average noise and synchronization can be understood by correlating the global signal with the behaviour of an individual in the audience. We demonstrated this by recording the local sound intensity in the vicinity of a group of oblivious individuals (Fig. 1b). In the incoherent phase, the local signal was periodic, with a short period corresponding to the fast clapping of an individual in the audience. However, the clapping period suddenly doubled at the start of the synchronized phase (at about 12 seconds in Fig. 1a,b), and slowly decreased as synchronization was lost (Fig. 1e).

The decrease in the average noise intensity is therefore a consequence of the period doubling, because there is less clapping per unit time. An increase in the average noise intensity is possible only by decreasing the clapping period, which can indeed occur (Fig. 1e). However, the decreasing clapping period gradually brings the synchronized clapping back to the fast clapping heard in the early asynchronous phase, and synchronization disappears. Apparently, this conflicting desire of the audience simultaneously to increase the average noise intensity and to maintain synchronization leads to the sequence of appearing and disappearing synchronized regimes.

These results indicate that the transition from random to synchronized clapping is accompanied by a period-doubling process. To determine whether period doubling is in fact a necessary condition for synchronization, we investigated the internal frequency of clapping by several individuals in controlled clapping experiments. Individual students, isolated in a room, were instructed to clap as they would immediately after a good performance (mode I clapping) or during the rhythmic applause (mode II clapping).

The frequencies of the two modes of clapping are clearly separated and the average period doubles from mode I to mode II clapping (Fig. 1f). Most important, however, is that the width of the frequency distribution and the relative dispersion of mode II clapping is considerably smaller, a result that is reproducible for a single individual as well (Fig. 1g).

Our results indicate that after an initial asynchronous phase, characterized by high-frequency clapping (mode I), individuals synchronize by eliminating every second beat, suddenly shifting to a clapping mode with a double period (mode II) where dispersion is smaller. For a group of globally coupled oscillators, the condition for synchronization is that dispersion must be smaller than a critical value. Consequently, period doubling emerges as a condition for synchronization, because it leads to slower clapping modes during which significantly smaller dispersion can be maintained.

Our measurements offer an insight into the mechanism of synchronized clapping: during fast clapping, synchronization is not possible owing to the large dispersion in the clapping frequencies. After period doubling, as mode II clapping with small dispersion appears, synchronization can be and is achieved. However, as the audience gradually decreases the period to enhance the average noise intensity, it slips back to the fast clapping mode with larger dispersion, destroying synchronization.

In summary, the individuals in the audience have to be aware that by doubling their clapping period they can achieve synchro-
Leptin and diabetes in lipoatrophic mice

Lipoatrophic (lipodystrophic) diabetes is a disorder in which insulin resistance and hyperglycaemia are associated with a reduced body-fat mass, in contrast to the usual association of diabetes with obesity. Transgenic mice with differing degrees of fat loss can be used as models for lipoatrophy. Using the p2-SREBP-1c mouse, which has a moderate fat deficiency, Shimomura et al. showed that leptin treatment reverses the diabetes, concluding that insulin resistance in congenital generalized lipodystrophy can be explained by a leptin deficiency. However, we have used a more severe model of lipodystrophy, the A-ZIP/F-1 mouse, in which we find that leptin treatment is only slightly effective in correcting diabetes.

A-ZIP/F-1 mice (Table 1) have an almost complete lack of white adipose (fat) tissue, a severe resistance to insulin, diabetes, and greatly reduced serum leptin levels. We found that infusing leptin into A-ZIP/F-1 mice at the same rate (5 μg day⁻¹ for 4 weeks, starting at 7 weeks of age) and to produce the same serum leptin level (3 ng ml⁻¹) as in Shimomura et al.’s mice had no effect on serum glucose or insulin concentrations (results not shown). A higher leptin dose (30 μg per day, causing leptin to rise by 5 ng ml⁻¹) did reduce glucose and insulin levels (Fig. 1), food intake (6.6±0.3 to 4.7±0.2 g day⁻¹, P<0.001), and liver weight (from 3.04±0.04 to 2.09±0.11 g, P<0.001). Even at this higher dose, however, our mice still had markedly raised blood glucose and insulin levels.

In our A-ZIP/F-1 mice, the efficacy of leptin treatment diminished with age: at 13 weeks (the age of Shimomura et al.’s mice), leptin had a minimal effect (Fig. 1), and no effect at all at 28 weeks (results not shown). In contrast, leptin infusion into 13-week-old leptin-deficient ob/ob mice completely normalized both glucose and insulin levels (Fig. 1).

Evidence from humans and mice supports the conclusion that leptin deficiency cannot completely explain the diabetic phenotype of generalized lipodystrophy. Patients with generalized lipodystrophy are more prone to diabetes than are those who lack leptin; similarly, A-ZIP/F-1 mice are more diabetic than ob/ob mice (Fig. 1). Thus, leptin deficiency contributes to the insulin resistance of generalized lipodystrophy, but is neither the sole nor the principal cause of insulin resistance in severe forms of this disease.

The observed differences between the A-ZIP/F-1 and a p2-SREBP-1c mice are probably due to their different amounts of fat, although transgene-specific effects or their different genetic backgrounds may play a part. A p2-SREBP-1c mice have more residual adipose tissue: in these mice, leptin appears to be limiting and its replacement reverses their diabetes. A-ZIP/F-1 mice must experience loss of other functions provided by adipose tissue besides leptin secretion — for example, functions that affect fatty-acid and triglyceride metabolism. Alternatively, adipose tissue might exert a direct or indirect endocrine effect.

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Shimomura et al. reply — Human lipodystrophy (also called lipoatrophic diabetes) is genetically heterogeneous, with the severity of insulin resistance and diabetes mellitus varying widely depending on the degree of reduction in adipose tissue mass and the age of the patient. It is therefore not surprising that two mouse models of lipodystrophy (created by using different transgenes, A-ZIP/F-1 and p2-SREBP-1c) vary in their disease severity and in their sensitivity to leptin. The p2-SREBP-1c animals respond to leptin with a decrease in their insulin and blood sugar levels, whereas the A-ZIP/F-1 animals of Gavrilova et al. apparently manifest leptin resistance. The differences between these two models should not preclude a clinical trial of leptin in leptin-deficient patients with lipodystrophy, with continuation of therapy in those who are leptin-sensitive.

Table 1 Differences in the severity of the A-ZIP/F-1 and p2-SREBP-1c phenotypes

<table>
<thead>
<tr>
<th>Phenotype</th>
<th>Epidydymal fat mass</th>
<th>Brown fat mass</th>
<th>Glucose (mg dl⁻¹, non-fasting)</th>
<th>Insulin (mg ml⁻¹, non-fasting)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A-ZIP/F-1</td>
<td>1</td>
<td>50</td>
<td>1,000</td>
<td>60</td>
</tr>
<tr>
<td>A-ZIP/F-1</td>
<td>30</td>
<td>400</td>
<td>300</td>
<td>20</td>
</tr>
<tr>
<td>p2-SREBP-1c, 30</td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tbody>
</table>

Data are for 7-week-old mice.