

PHONONS AND LONG-RANGE COHERENCE IN CELLS

J.S. GOMEZ-JERIA

*Departamento de Química, Universidad de Chile,
Facultad de Ciencias Básicas y Farmacéuticas, Casilla 653, Santiago, Chile.*

Received: 16 June 1982

It has been proposed that a branch of collective longitudinal electric modes exist in many biological systems⁽¹⁾. The frequencies of the vibrations lie in the microwave region (10^{11} – 10^{12} Hz). The dipolar oscillations of biological structures (that is, H-bonds, cell membrane, pockets of non-localized electrons), will be coupled through long-range Coulomb interactions. The models in which these predictions were made have received theoretical treatment^(2,3), and Cooper has developed it making emphasis in the cell cycle and in the cancerous state^(4,5).

In this letter we would like to comment on three questions which are of relevance in a realistic model for the cell. (a) What may be the principal phonon sources in cells? (b) Is there any biological structure in the cell that may be a relatively stable source for electromagnetic waves? (c) If the phonon sources in cells are important, what are the possible biological effects of this fact?

In relation to the first question, a survey of protein structure shows that about 90% of all internal polar groups form H-bonds, a fact consistent with the large amount of secondary structure observed in them⁽⁶⁾. Also, if we consider that the protein-ligand interactions involve non-covalent forces (that is, H-bonds, dispersion forces, etc.), we can see that inside the cell, the number of H-bonds is very great. Considering that H-bonds absorb in the microwave and far infrared region regions⁽⁷⁾, we believe that they are one of the most important sources of long-wave phonons in cells.

The second question calls for some reflections. If there is a stable branch of collective longitudinal modes in the cell, we may accept as a working hypothesis that in order to produce any important biological effects, the branch must be stable during the most part of the cell cycle at least. On the basis of this hypothesis we suggest that the cell's membrane is the structure that produces a stable electromagnetic field inside the cell through its vibrations. It is interesting to mention that the cell's membrane area increases during the cell cycle, modifying the strength of the field inside the cell. On the other side, there is a very important source of long-wave phonons that is stable during most of the cell cycle: the nucleoprotein (DNA plus its associated proteins).

The coupling of the electric field with these phonons will produce phonon-like polaritons.

The third question is more difficult to answer. From Bose-Einstein statistics and thermodynamic considerations, it follows that the phonon entropy corresponding to the quantity of phonons produced by one mole of harmonic oscillators with frequency ν is:

$$S(\nu) = \frac{N h \nu}{T} n(\nu) - N k \ln \left(\exp \frac{h \nu}{k T} \right) \quad (1)$$

where h , k , N , T and $n(\nu)$ are the Planck's constant, the Boltzmann's constant, the Avogadro's number, the absolute temperature and the number of phonons in energy level $h\nu$, respectively.

We see that for a given temperature, $S(\nu)$ depends only on the frequency of the oscillator. In the case of a covalent bond, the frequency of vibration lies on the range $2.5 \cdot 10^{13} - 5.0 \cdot 10^{14} \text{ sec}^{-1}$ and the $S(\nu)$ value is small (between 0.15 and 10^{-7} cal/mole $^{\circ}\text{K}$). But the case of H-bonds is very different, the decrease of ν leading to relatively high $S(\nu)$ values. Having $\nu = 1.5 \cdot 10^{12} \text{ sec}^{-1}$ we obtain $S(\nu) = 4.8$ cal/mole $^{\circ}\text{K}$. Therefore, these phonons can play a very important role in the process of protein-ligand interactions. For example, at absolute temperature of 298°K , we obtain for $T\Delta S$ a value of 1.430 Kcal/mole, a value that is important for the determination of the free energy of binding in enzyme reactions. Perhaps the consideration of phonon entropy may resolve small discrepancies observed in the values of the free energy of binding in chymotrypsin inhibitors⁽⁸⁾.

Work on mathematical formulation of these facts is in progress.

Acknowledgment

I thank Prof. Dr. M. Castillo for helpful comments.

References

1. Frohlich, H., in *Theoretical Physics and Biology*, ed Marois, p. 13, North-Holland (1969).
2. Frohlich, H., *J. Collective Phen.*, 1, 101-109 (1973).
3. Frohlich, H., *Int. J. Quant. Chem.*, 2, 641-649 (1968).
4. Cooper, M.S., *Phys. Lett.*, 65A, 71-73 (1978).
5. Cooper, M.S., Presented at the International Workshop of Physical Concepts in Tissue Growth, Bad Neuenahr, RFA (1979).
6. Chotia, C., *Nature*, 248, 338-339 (1974).
7. Pimentel, G. and McClellan, A., *The Hydrogen Bond*, W.H. Freeman and Co., San Francisco (1960).
8. Christova, E., Yomtova, V. and Blagoev, B., *Int. J. Peptide Protein Res.*, 15, 459-463 (1980).