Experimental

MECHANISMS RELATED TO CHARGE DENSITY PULSE FORMATION IN LIVING SYSTEMS

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Abstract

In this paper we discuss empirical approaches designed to elucidate the mechanisms involved in the formation of Charge Density Pulses (CDP) originating in living organisms. These oscillating pulses originate intracellularly in an unstable, dissipative system and are manifested as interfacial reactions located at the tissue-metal electrode contact zone. In both plants and animals the characteristics of the input energy are consistent with intracellular homeostatic mechanisms which dissipate in accordance with a precise log-time function. Studies with living systems support a conjecture of CDP specificity within tissue types; showing that the most active oscillations occur in those tissues with higher metabolic rates. In human subjects, injury and its associated pain, produces significant alterations in the CDP waveform, the magnitude of which can be utilized to quantitatively determine pain levels. Specific examples of applying CDP methods in trauma and pain monitoring are given in Levengood and Gedye, U.S. patent No. 6,347,238 B1, issued February 12, 2002.[1]

From the application of reaction rate theory the Gibbs free energy of activation, E_a for CDP formation in *Dacus carota* roots was found to be 9.4 kcal/mole. In addition, strong support for enzymatic control of the CDP oscillatory process was provided by the observation that the dissipative rate constant (k-value) dropped very sharply as the tissue temperature reached 40°C, a critical temperature which in most plant species is well known to produce enzymatic deactivation and inhibition of respiration. From these enzymatic studies as well as from data obtained in the early phases of our work, it became quite clear that CDP pulses are composed of charge carrier mechanisms with properties far more complex than those observed under conditions of classical electronic conduction. The observed non-ohmic conductivity patterns are suggestive of the kinds of superconductivity mechanisms found in Josephson Junction systems where charge carriers are formed within metal-metal oxide layers.

KEYWORDS: Charge density pulses, living systems, energy transport

INTRODUCTION

The presence of organized "electrodynamic fields" around living systems was first brought to the attention of the scientific community by Professor Harold Saxton Burr, and the results of his prolific research studies involving this energy, are discussed in his 1972 book, ***Blueprint for Immortality***.[2] Burr's active research concerning these electrodynamic fields was conducted in the interval from the mid 1930's to the mid 1950's, at a period just before the advent of more sophisticated, sensitive and computerized devices for monitoring very subtle variations in charge transport within living systems.

In the research results presented here we demonstrated that these electrodynamic fields are the result of what we define as Charge Density Pulses (CDP), which originate within the cells of the organism. Although the main purpose of this paper is directed toward elucidating those mechanisms related to the formation and distribution of these CDP pulses, we have been asked by the reviewers to make its relevance to energy medicine more clear. Therefore we wish to point out that in the case of human subjects, specific characteristics of the CDP waveform may be utilized to quantitatively determine pain levels. Examples of its use in pain monitoring are presented in Levengood and Gedye, U.S. Patent 6,347,238 B1, issued February 12, 2002.[1] Recent analyses of CDP waveforms have shown that other characteristics of the response patterns can be correlated with psychological, emotional and environmentally interactive states of test subjects. Details will be presented at a later date.

The pattern of charge distribution and the direction of current flow in a Charge Density Pulse (CDP) originating within living systems is determined by the characteristics of the input energy originating within the living tissue. This charge carrier energy has properties consistent with an unstable, dissipative system with persistent fine structure oscillations. By applying perturbation kinetics we have shown that these oscillating CDP waveforms originate within the cells and are related to membrane transport mechanisms.[3] Although the origins of the pulses are internal, one must also consider the overall systematic organization of energy transport between the electron interactions originating at the cellular level and the final energy transfer which takes place as an interfacial reaction at the tissue-electrode contact zone. The CDP waveform as well as its polarity, is established at the tissue-metal electrode contact zones. This general type of interfacial reaction was discussed in a recent review where it

was pointed out that charge transfer in interfacial reactions is an extremely important process in a variety of systems; it is involved in mechanisms ranging from photosynthesis and respiration to electrochemical energy systems.[4]

Energetically, the maximum amplitudes (P_a) of the current pulses developed at the electrode-tissue interfaces are observed to lie in the region of 1 to 10 microamps, a range which is consistent with bioelectric currents developed during normal membrane transport processes in living systems. Since we find that the basic CDP patterns, responses to external perturbations and polarity differences are similar in both plant and animal tissues we make the provisional working assumption that the basic mechanisms of charge density pulse formation are of a similar nature in both tissue types. It will be demonstrated that this is a reasonable assumption. By accepting and examining the properties of this putative reciprocity model we are able to put forth possible interrelationships between the known, fundamental electrokinetic mechanisms in living cells and the electrophysiology involved in the CDP phenomena.

In the final phase of our tentative model building we examine details of modifications in the form of the sustained CDP oscillations which are brought about by interposing various metals between the living tissue and the aluminum electrodes. Within this section we also discuss the implications of the earlier, surprising finding that charge transport through the interposed metals takes place under non-ohmic conditions analogous to those found in superconducting charge transport processes.

EXPERIMENTAL METHODS

The experimental methods for producing the CDP traces are basically similar to those described in our original publication.[3] Since the procedure for obtaining the pulses from human hands is somewhat different from that employed in the examination of living plant tissues, both of the procedures are described separately as follows.

CDP—Human Hand Contact

The CDP responses from human hands were generated by placing the palms of the hands on the outside surface of parallel, vertically positioned, semi-

polished aluminum plates. These circular, charge collector electrodes are 31 cm diameter, about 0.6 cm thick and separated by four nylon spacers which provide an 8 cm air gap. Voltage fluctuations were detected by placing a 1.0 Kohm resistor across the electrodes. Lead connectors at the edges of the plates were machined about one cm deep into the base metal. Additional leads, soldered to each side of the resistor, extended to the input of a millivolt chart recorder with a maximum full range sensitivity of 1.0 mv and a linear frequency response of about 2 Hz. With most test subjects the recorder sensitivity was set at 10 mv full scale.

CDP—Living Plant Tissue

The aluminum contact electrodes used in the plant investigations consisted of two 0.6 cm diameter rods 12.5 cm long, with a lead wire welded to one end of each rod. The opposite, tissue contact ends were machined flat and given a semi-polished surface. As in the hand contact method the lead wires were connected to each side of a 1.0 Kohm resistor and extended to the chart recorder.

The studies reported here were conducted with living plant stem sections or root tissue mounted on a non-conducting base and with the longitudinal axis in the horizontal position. The rod designated as cathode (by its polarity sign going into the chart recorder) was placed in contact with the basipetal (root) end of the plant tissue, and the anode in contact with the acropetal (shoot) end. The contact pressure was sufficient to secure the tissue section between the electrodes without crushing the plant cells. The rod contact area was 0.28 cm^2 in tissue sections 0.6 cm or larger. With the commonly observed P_a values of around 0.5 microamps the current densities lie in the range of 1.8 microamps per cm^2.

The CDP trace shown in Figure 1 is typical of those obtained from both human hands and plant tissue. These chart data were analyzed by recording the peak amplitude (plus or minus) at the point on the curve where the current began to drop back to the base line level. This inflection point (designated as P_a) occurs well after any initial contact potential changes, usually 2-10 sec. into the trace. The value of P_a as given in this paper is in microamps. This peak

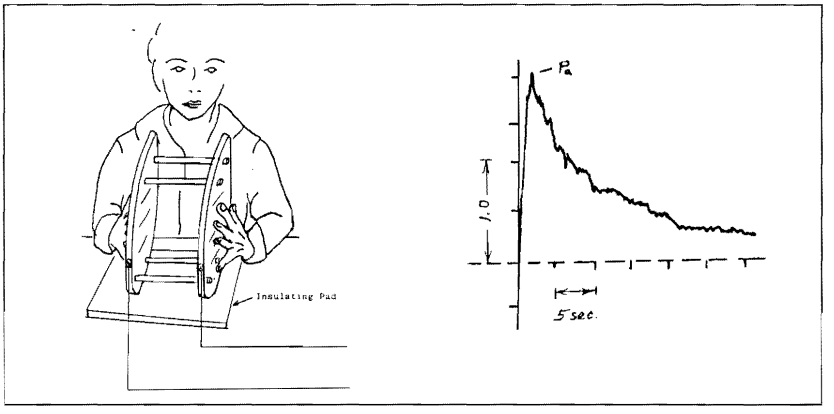

Figure 1. *Charge density pulse (CDP) system depicting hand placement on the electrodes; right is a typical 30 sec. hand trace showing the dissipation with time (P_a indicates peak amplitude).*

amplitude analysis provides information at one time point in the response curve. In a later section we discuss the specific form of the response curves. The maximum current density I_d (microamps/cm^2) through the tissue is given by,

$$I_d = P_a/a_t \qquad (1)$$

where a_t is the area of tissue contact and in adult humans is in the range of 90-110 cm^2.

CDP Formation at the Cellular Level

When a living system is exposed to the CDP plates an immediate readjustment takes place in the bioelectric fields of the organism. It is presumed that this is manifested at the cellular level as rapid changes in membrane potentials. Therefore, if we expect to understand the origins of these unstable, oscillating charge groups we must consider the relationship between ionophoretic membrane transport and the kinetics of intracellular electric field patterns and interfacial interactions. In plants exposed to an electric field directed at an angle θ, the electrophoretic migration pattern of charged particles (such as chloroplasts) onto the surface of the nuclear membrane, has been shown to follow the theoretical relationship,[5]

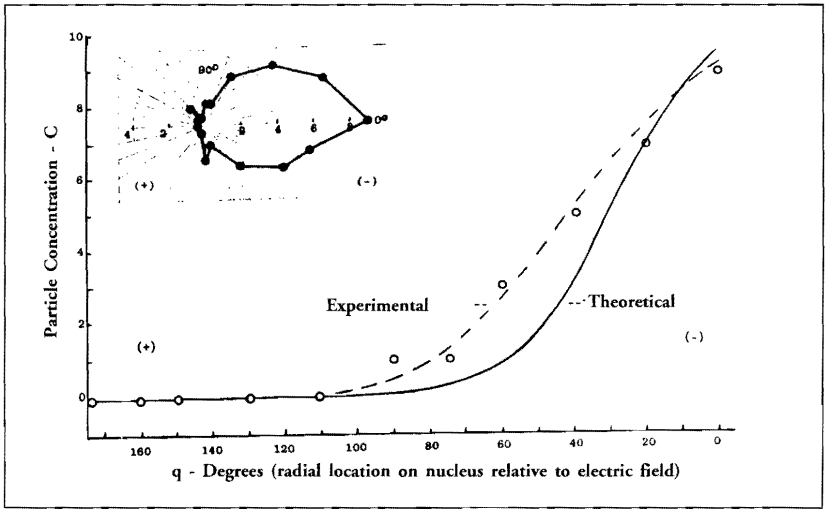

Figure 2. *Distribution of submicrometer particles around a nucleus in an electrophoretic field. Comparison of experimental data with theory (see text). Insert shows the radial concentration of particles around the nucleus.*[5]

$$n = n_a [\sigma \bullet \text{csch}(\sigma)] \exp [\sigma \bullet \cos(\theta)] \quad (2)$$

where n_a is the average particle density, csch is the hyperbolic cosecant and σ is related to the electric field by

$$\sigma = (u/D)(V_p/2) \quad (3)$$

where u is the ionophoretic mobility, D is the diffusion constant and V_p is the potential difference produced by the electrophoretic field conditions which without an externally applied field would be governed by cell membrane potential and metabolically driven ion currents in the cytoplasm. Equation (2) predicts a crescent outline in the particle distribution and as shown by the empirical data presented in Figure 2 (reproduced from Figure 2 of reference 5), the chloroplast concentration profile follows the theoretical relationship very closely. In biophysics, or for that matter in science in general, mathematics is used to represent the attributes of a system; it is therefore, highly relevant to this discussion to point out that this same crescent distribution pattern has been observed in the electrophoretic migration of fluorescent-labeled receptors

on the surface of spherically shaped basophilic cells in rat tissues.[6] In other words we observe here mathematical and empirical confirmation of correspondence between the electrophoretic mobility patterns in both plant and animal tissue.

These induced cellular membrane fields can also influence the electrokinetic movements of the internal nucleoli; manifested as displacements from their quasi-equilibrium position. In plants the nucleoli possess a net negative dipole charge.[5] When d is the distance (in micrometers) from the nuclear membrane to the nucleolus, the migration function is given by,

$$d = d_o e^{-at} \qquad (4)$$

where d_o is the separation at $t = 0$ min. and a is a constant. Under an externally applied field strength of $v_p = 4\ v\ cm^{-1}$, the migration rate is in the order of 0.1 micrometers per minute. These kinetic studies in plants demonstrate that any induced change in the spatial configuration of organelles, either within the nucleus or within the cytoplasm would be expected to produce changes in the free charge distributions within the nuclear and cell wall membranes.

Separations of ionic species across cell wall membranes normally generate field strengths in the range of $10^4\ v\ cm^{-1}$, but do not present a serious problem for space charge limitation of current flow through biological systems, and as shown in Figure 2, do not completely shield the nucleus or the nucleoli (Equation 4) from applied electrophoretic fields.[7] A direct association between cellular metabolism involved in the growth of root primordia in bean hypocotyls and the degree of electrophoretically induced nucleus-organelle-clustering has been reported.[8] Fluctuations in the membrane-organelle associations could also explain periodic changes in the electrophoretic mobility of *Chlorella* cells within a *synchronized* culture.[9]

Since metabolic activity can be linked with electrophoretic processes in cells any sustained metabolic oscillation would be expected to induce fluctuations in the distribution of free charge carriers in the cell membrane and thus produce oscillations in the external electric field which can be experimentally detected as CDP waveforms.[8] Furthermore, within one *specific tissue region* which we will designate here as tissue "type A," the metabolic oscillations or enzymatic

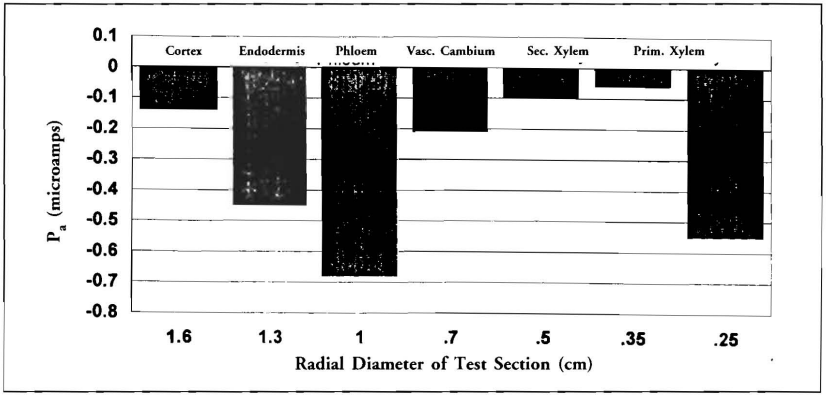

Figure 3. *Peak amplitude (P_a) levels in carrot root tissues. Minimum probe electrodes applied transectionally.*

activity within each cell would be expected to be in synchrony, therefore the effect could be additive from cell to cell (like voltaic cells in series) with the result that relatively large amplitude, oscillating, trans-membrane currents would be formed.

Within a neighboring, contiguous zone containing tissue "type-B" functioning with entirely different metabolic pathways, the direction of current flow, or what could be considered charge density "polarity" may be opposite that in the type-A tissue. The amplitudes and frequencies of the pulses originating in the type-B tissue would be expected to have completely different waveform characteristics compared with those originating in the type-A tissue; as a consequence both destructive and constructive interference effects could take place. In preliminary computer studies relating to the fine structure characteristics within the CDP traces, we find definite indications of the expected resonance effects.

Furthermore, studies with living carrot roots support the above hypothesis of CDP specificity in tissue types. In these tests the aluminum probes were positioned to obtain transectional CDP traces across a carrot root in which various tissue regions were sequentially exposed. The results summarized in Figure 3 show that the most active CDP oscillations occur in the phloem (food transport tissue) and the primary xylem (water transport tissue). The transition type tissues display low to intermediate P_a levels.

INTERACTIVE REACTION DYNAMICS AT THE ELECTRODE-TISSUE INTERFACE

Under steady-state dissipative conditions Nicolis and Prigogine point out that cellular metabolism can itself generate sustained oscillations which "are far from exceptional in living cells."[10] The empirical data obtained in a typical CDP trace are in complete agreement with this reference to patterns of sustained oscillations. After reaching a peak amplitude P_a the envelope of a CDP curve decreases non-linearly toward the base level, the general form of which is given by,

$$I = -k[\ln(t)] + b \tag{5}$$

where I is the current level at t sec. into the trace, k is the rate constant and b the intercept. This relationship is suggestive of an unstable system in which the current density exhibits a systematic decrease with time. During a trace interval we will let the rate of charge density pulse dissipation be a function of P_a, t and u_p as follows,

$$-dI/dt = f(P_a, t, u_p) \tag{6}$$

where P_a is the maximum current level at the inflection point in the CDP trace and u_p the ionic mobility of the charge pulses. The current is taken as inversely proportional to time since the level is dependent on a limited "reservoir" of membrane reaction sites. From this we have the rate function,

$$dI/dt = -(P_a u_p)(1/t) \tag{7}$$

and

$$\int dI = -(P_a u_p) \int (1/t)dt \tag{8}$$

which after integration gives,

$$I = -(P_a u_p)[\ln(t)] + b' \tag{9}$$

since for any given trace P_a and u_p are constant, this rate function is identical in form with Equation (5) obtained from empirical data.

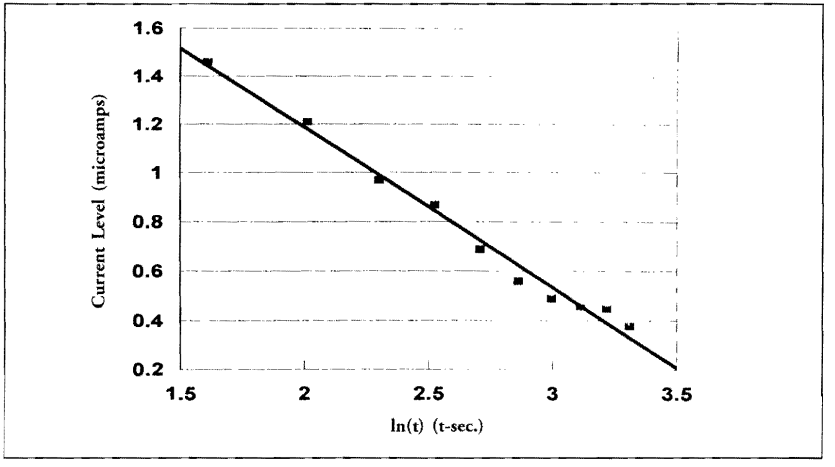

Figure 4. *Data from a typical CDP dissipation curve showing the relationship between current level and ln-time; R^2 = 0.98 for linear regression curve (data from WCL, 8:00am, 7-3-00).*

The very systematic decrease in the CDP current level as a function of log-time may be considered in relation to the kinetics of homeostasis operating within perturbed metabolic pathways. In this system the perturbing energy in the CDP trace originates within the initial interactive electrokinetics which take place between the biofield of the organism and the aluminum metal of the electrode plates.

In Figure 4 there are data from a typical CDP-hand trace plotted according to the Equation (9) rate function. The individual data points in Figure 4 are not randomly distributed along the regression curve, but rather, seem to oscillate about the regression line in a manner typical of a feedback loop mechanism. A system of this kind gives rise to a mixture of regularity and disorder, it is definitely not a completely random process. This perturbed wave complex system appears to form organized ion-electron pulses which drift back and forth through the organism between the points of contact at the electrode plates and with temporally decreasing amplitudes which end up near, but not at the base or zero level.

As we have pointed out above—from the empirical evidence it seems quite clear that the CDP patterns are associated with trans-membrane electric fields,

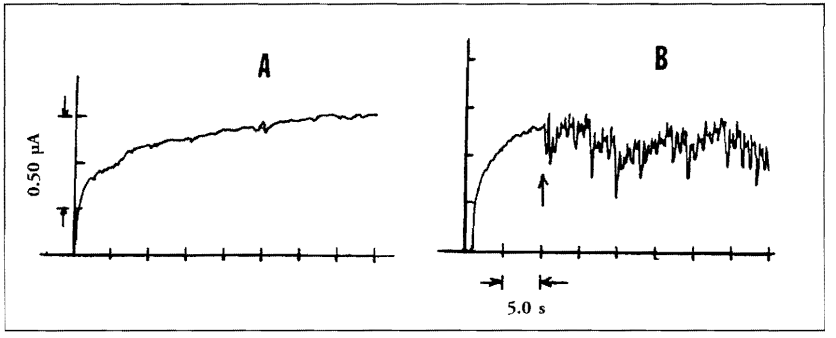

Figure 5. *CDP waveforms obtained with brass probe cathode (see text) placed on the pectoral region; A-trace held stationary and B-trace oscillated in a lateral motion over a distance of about one cm and at one cycle per second (right hand on the anode aluminum plate, see Figure 1).*

where as stated by Gutmann the formation of solitons may take place on a cell membrane as an energy transductive step.[7] This transductive concept has been discussed in a theoretical model which suggests that a mechanical signal at the cell membrane surface can be transduced into an electromagnetic wave in a self-consistent manner characteristic of soliton wave formation.[11]

We examined this proposed intracellular, mechanical transduction concept in the following manner. A brass probe, identical in design to the probes discussed in the Methods section, was attached to the cathode side of the recorder (insulated lead wire about 2 meters long). A probe test was conducted by placing the right hand on the anode plate (Figure 1). While holding the electrically insulated handle, the brass probe was placed in contact with the area of skin selected for study.

Under normal conditions it is possible to mechanically displace the skin at most regions on the human body. With lateral displacements (left or right) in the range of 1-cm, repeated cycles can be applied with little or no discomfort. For this series of experiments the pectoral region was chosen because it offered a relatively flat area in which uniform skin contact could be maintained across the surface of the probe. The CDP trace in Figure 5a was obtained with the probe held stationary in the central region of the right pectoral (WCL). In Figure 5b the probe was applied to the same region and after 10 sec. into the trace, it was oscillated in a lateral motion producing

a 1 cm left stroke followed by a 1 cm right stroke, at a rate of around one complete cycle per second. Each mechanical shearing cycle produced a CDP pulse having an amplitude far exceeding the fluctuations observed in the trace with the probe held stationary. By applying this "portable" charge collector electrode in a mechanical shearing mode, we observe clear indications that the proposed transduction mechanisms do indeed involve ion-gating characteristics.

Within a data base consisting of over 5000 CDP traces (both humans and plants) we have never observed the absence of the dissipative oscillations or a trace pattern in which the small amplitude pulses disappear and the trace "flat lines" along the zero base line. The ubiquitous nature of the low amplitude pulses suggests that their maintenance depends on active transport mechanisms which direct homeostasis during the interval of biofield perturbation (tissue contact). If the proposed active transport of CDP pulses involves simple enzyme kinetics, the general effect of temperature on the reaction rates may be analyzed through the use of the Arrhenius relationship given by,

$$k = A[\exp.(-E_a/\mathbf{k}T)] \qquad (10)$$

where k is the reaction rate constant which in a CDP trace is equivalent to the dissipation constant given in Equation (5); the term E_a is the Gibbs free energy of activation, $\boldsymbol{k}$ the Boltzmann constant and T absolute temperature. By taking the logarithm of both sides of equation (10) we obtain the more useful expression,

$$\ln(k) = -E_a(1/\mathbf{k}T) + \ln A \qquad (11)$$

from which we can empirically determine the slope constant E, the activation energy of the system.

An essentially equivalent form of the Arrhenius equation has been successfully applied to many systems involving the thermal statistics of self organized electron transfer processes.[4] In interfacial reactions the surrounding medium, in the present case the living tissue, has a crucial effect on the interfacial electron transfer. To examine the enzyme kinetic aspect of our model we used the roots from living carrot (*Dacus carota*) plants. Root sections 4-5 cm dia. and 4 cm

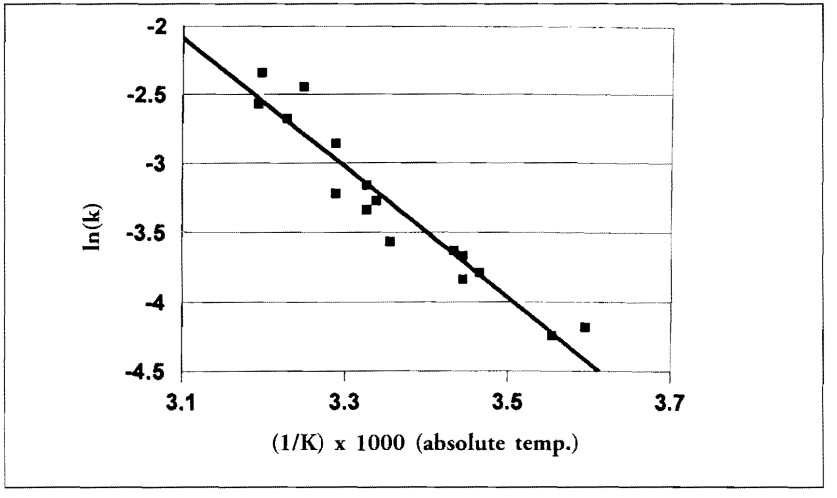

Figure 6. *Arrhenius plot of CDP dissipation rate constants (k) obtained from the primary xylem in carrot tissue (longitudinal probe placement along 5 cm sections).*

long were excised from the apical end, wrapped in Saran plastic then sealed in a plastic bag. This sealed sample bag assembly was lowered into a temperature controlled water bath and allowed to reach temperature equilibrium (around 30 min..). After removal from the water bath a CDP trace was taken along the center line of the root. The 6 mm diameter, aluminum probe electrodes were placed in the zone of primary xylem (see Figure 2), with the cathode contacting the basipetal end of the sample section.

From each CDP-temperature trace the rate constant k was determined from linear regression analysis applied to time-normalized data plotted according to the Equation (5) relationship. A total of 16 tests were conducted over a temperature range of 5° to 39°C. These data are plotted in Figure 6 according to Equation (10); the high correlation coefficient of $r = 0.96$ indicates that temperature activated reactions are involved and from the slope we obtain an activation energy of 9.4 kcal/mole. This value is somewhat lower than that found for most chemical reactions, and suggests the possibility that, in this case, the energy barrier to the activated state may be considerably reduced by intercellular soliton formation in self-organized electron-phonon associations.

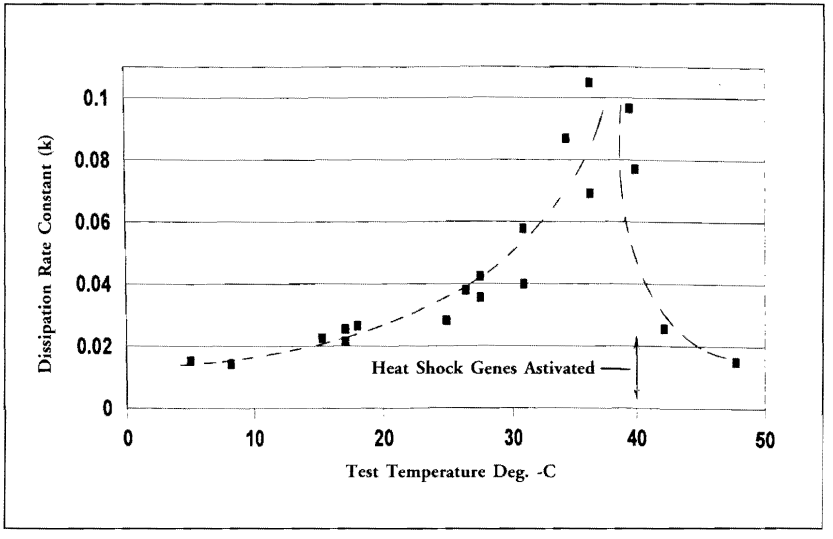

Figure 7. *Variation in CDP dissipation rates from carrots heated into the "heat shock gene" activation region.*

An additional factor providing strong support for enzymatic control of the CDP oscillatory process was the observation that the rate constant *k* drops very sharply as the tissue temperature reached 40°C. Shown in Figure 7 are *k* values obtained from individual tests conducted both below and above the critical 40° point. In the region of 40°-45° most of the normally functioning enzyme systems are deactivated and in this same temperature region heat shock genes are activated. In most plant species this produces the inhibition of respiration.[12] Since secondary alterations are produced in the enzymatic reactants the Arrhenius reaction rate model is no longer applicable.

CDP INTERACTIONS WITHIN THE ELECTRODES

The specific details of oscillatory patterns observed in CDP dissipation traces (as shown specifically in Figure 5) are defined by conditions at the tissue-metal interface zone. The bioelectric energy field in the tissue *interacts* with the metal lattice of the electrode plates to produce the charge transfer current. In

most non-biological electrical circuits operating at room temperature the primary carriers of the current are high mobility electrons and to some extent slower moving, positive ion holes and vacancy defects. It became quite clear from data obtained in the early phases of our work that CDP pulses are composed of charge carrier mechanisms with properties far more complex than those observed under conditions of classical electronic conduction.

Deviations from the usual conductivity patterns were observed in simple experiments in which small samples of aluminum and copper, with quite diverse cross sectional shapes, were interposed between the palms of the hands and the electrode plates.[1] From the classical view of electronic conduction in metals the level of P_a should be directly related to the area-length ratio (A/L) for each inserted test object; however, our findings (Figure 7 or Reference 3) disclosed a relationship with the cross sectional area (A) of the interposed metal and essentially independent of the conductivity path length (L). This type of conductivity is highly suggestive of charge transport taking place within a metal lattice in which soliton wave formation occurs with minimal loss of linear energy propagation.

In aluminum oxide (Al_20_3) the energy band gap for electron conduction is 8.3 ev, whereas in copper oxide Cu_2O it is 2.2 ev, and from this one would predict that the charge density amplification of P_a in copper, applied as an insert metal, would greatly exceed the levels found in the aluminum inter layers.[13] With copper inserts examined in the same manner as the aluminum (Table I, Reference 1) the P_a currents were increased from 3 to 30 times the P_a levels found with the aluminum inserts.

Although the P_a levels were increased with the copper inserts, another notable feature of the CDP traces was a marked reduction in the amplitudes of the oscillations in the dissipative curves relative to those found with the aluminum inserts. This suggests that the biofield potentials are inducing unusual electrical conduction mechanisms in the metals, with the current levels P_a and the amplitudes of the dissipative oscillations being related to the electron energy band gap as modified by the accompanying oxide layer. Fundamentally this experimental arrangement may be thought of as a crude Josephson junction, where the conductive elements are each separated by a thin, oxide film.[14] The conduction of charge carriers across interfaces between the metal-oxide layers in a Josephson junction deviates significantly from ohmic conductivity.

Table I
Surface Plasmon Energies in Various Solids*

Solid	Surface Plasmon Energy
Aluminum	8.8 ev
Copper	3.5 ev
Silver	3.5 ev
Gold	2.5 ev
Magnesium	6.3 ev
Iron	5.0 ev

*data from reference 15, p. 379.

Since all living organisms emit electromagnetic radiation (primarily in the infrared region) it seemed worthwhile to consider possible photon-bioelectric field interactions at the interface regions between the living tissue, oxide layers and metal electrode. In a solid, a photon may give up its energy and create a phonon (or quantized lattice wave). Within the metal lattice the phonons may combine with free electrons to form e-phonon waves. In fact, a photon absorbed in matter gives up its energy and momentum to produce several other types of quantum excitations, each having its own quantized energy and momentum.

Photon absorption on the surface of metals can create quantized plasma (charge density) waves. These plasma energies are lower in the alkali metals with only one free electron per atom, than in those metals such as aluminum with divalent free-electrons.[15] Table I provides a list of surface plasma energies in various metals, several of which have been examined in our CDP studies.

Now if we compare the surface plasmon wave energies in aluminum with the wave energy in copper we find excellent agreement with the empirical findings. That is, if we assume plasmon wave formation in the charge density pulses originating at the tissue-metal contact zone we would predict that aluminum inserts would produce pulses with amplitudes in the range of 2.5 times (8.8/3.5) those produced with copper inserts. As mentioned in an earlier section, the aluminum insert traces with human hands produce oscillations in the range of 2.5 to 5 times the amplitudes in CDP traces with copper inserts.

The fact that the wave amplitudes in aluminum are somewhat higher than predicted by the (Al/Cu) ratio may be due to wave coupling or quantum phase interactions between plasmons-(e-phonons)-excitons (electron-hole pair) etc., each propagating within the oscillating electric field produced by the living tissue.

When we examined the peak amplitude levels (P_a) in the metal insert tests, we found that P_a is much higher with copper inserts than with aluminum, in other words, compared with the oscillatory effects, we have just the opposite situation. However, this difference is readily explained if we consider the fact that P_a relates to the total energy transferred through the *bulk* of the metal, whereas, the formation of the oscillating dissipation pulses refers primarily to quantum mechanical *surface* interactions. For charge transfer through the aluminum oxide film the energy band is 8.3 ev compared with 2.2 ev in the copper oxide film; therefore, the total charge transfer would be expected to be higher in copper with its lower band gap energy, again confirmed by the empirical data.

At the living tissue-bulk metal interfaces we appear to have molecular events activated by quantum interactions between photons emitted by the living tissue and the metal oxide matrix. Another unique feature of this *activated complex* is the oscillatory nature of these waveforms which travel through the bulk metal. In an earlier section we presented arguments relating to the fact that cellular metabolism can generate sustained oscillations in the bioelectric field. These biofield fluctuations in turn regulate the structure (oscillatory nature) of the waveforms organized at the interface region.

It is quite apparent from the consistent form of the dissipation function (Equation 5) that the reaction kinetics within the bulk metal matrix is not a random process. If this is, as indicated by Equation (5), an electrochemical rate process, then one would expect the bulk metal rate constant k_m to be dependent on temperature according to the Arrhenius relationship (Equation 10). In this situation we are interested in examining the electron reaction dynamics *within* the matrix of the aluminum electrodes. For this purpose we examined data from routine hand trace curves. Here, the primary temperature fluctuations take place in the metal and are related to ambient temperature conditions in the laboratory.

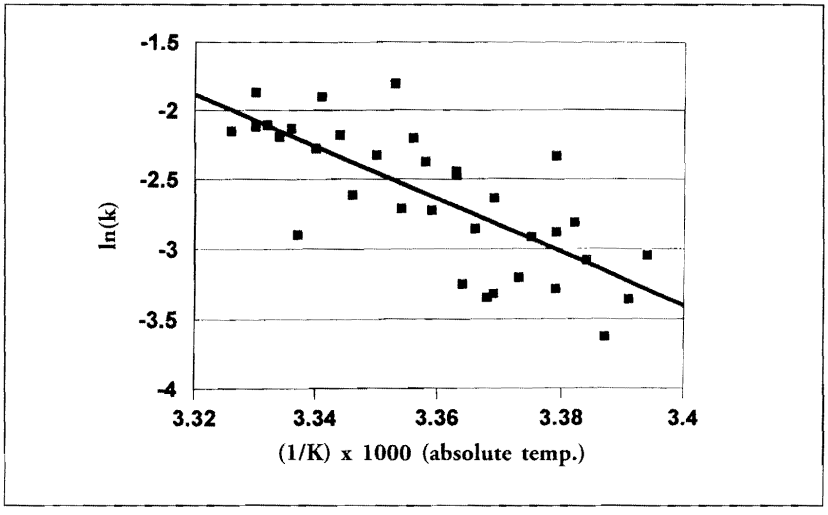

Figure 8. *Arrhenius plot of rate constants obtained from hand traces. Temperature indicated is that of the aluminum plate electrodes.*

Since the rate constant obtained from the hand trace dissipation curves can vary with the time of day, the season of the year and with various other phenological factors, the temperature data utilized here were taken from daily traces on a single subject obtained during the 7:00a.m. to 11:30a.m. interval during April and May, 1998, and covering a temperature range from 21° to 27°C. In an individual CDP trace, six data points were taken, starting at the P_a level (normalized at 5 sec. into the trace) and continuing every 5 sec. thereafter. For each CDP trace the current level was examined as a function of ln (t) and the value of k_m determined from linear regression analysis. The consistent form of the CDP dissipation function (Equation 5) was evident from the fact that the R^2 value from 35, I-log (t) curves were all in the range from (0.90 to 0.99).

The Arrhenius plot of these 35 CDP traces is shown in Figure 8 (r = 0.76), and from the slope we obtain a value for E_a of 37.8 kcal/mole. The activation energies of most chemical reactions lie in the region of 20-40 kcal/mole, therefore, the E_a for the CDP wave activation is well within the range of electrochemically activated complexes. In terms of charge transport energies

E_a = 1.64 ev, a value much lower than would be expected if the energy for charge transfer had to overcome the band gap energy for electron conduction in aluminum oxide (8.3 ev). If, however, quantum wave tunneling occurred within the plasmon and phonon waveforms, a much lower activation would be required. In this case photon energies might act as a "catalyst" and reduce the activation energy of the transition state complex.

For all liquids and solids, there is a UV frequency where light is strongly absorbed because the photon energy matches energy differences between filled and empty electron energy levels. Since, in living tissue there is a continuum of both empty and filled energy bands one might expect an extensive region of high energy absorption and photon storage.[16] If absorbed ultraviolet photons produce e-phonon or plasmon waves within the living system, one would anticipate that the CDP waveforms obtained from UV exposed tissue would display oscillations with higher frequencies and amplitudes when compared with the non-exposed tissue.

This hypothesis was examined by exposing the palms of the hands to a low intensity (5 watt) UV tube. The influence of a one minute UV exposure is shown in Figure 9 where the a-trace was taken before the exposure and the b, c and d dissipation curves were taken at 3, 10 and 30 min.. after exposure. In this test sequence there is a very pronounced increase in both the frequency and amplitudes of the oscillations in the CDP dissipation curves at the 3 and 10 min.. tests. At 30 min.. the UV induced perturbations are no longer evident. We can be quite sure that the UV influence is not due to a mechanical or thermal alteration in the stratum corneum. In our earlier work (Figure 6, Reference 1) the epidermal layers on the palms of the hands were severely abraded with a grinding tool. Although there was an increase in P_a immediately after abrasion (due to frictional heating) there were no induced oscillations with significantly higher amplitudes or frequencies.

The spectral emission characteristics of the "BLB" lamp used in these hand exposures has a single, very sharp emission band at 0.405 micrometers—it incorporates a glass envelope filter which cuts out most of the visible lines of the mercury spectra. With its single emission band this source provided a certain degree of coherence—which increased the probability that coherent phonons were generated within the intertissue regions of the hands, rather than the UV

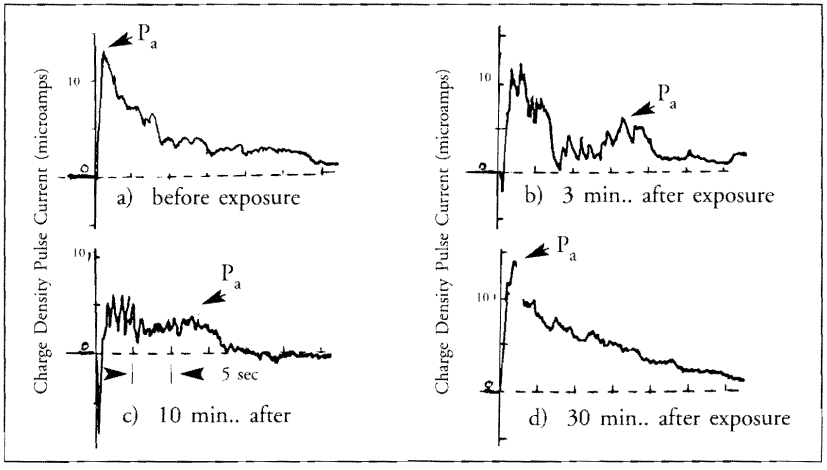

Figure 9. *Recorder chart traces showing CDP dissipation curves obtained by exposing the palms of the hands to a UV tube (hands held about 2 cm from tube during a 1 min.. exposure).*

energy becoming thermalized. Thus we are left with the possibility that solitons are produced by a dynamically stable interaction between an electron and a UV produced phonon—"a vibrational soliton." This system is far from equilibrium and the pronounced oscillations in the CDP dissipation curves such as shown in Figure 9 are very indicative of feedback in the UV perturbed tissue.

SUMMARY AND DISCUSSION OF CDP MODEL

Here, we have shown that contact between living tissue and the CDP electrodes produces an immediate readjustment of the total interacting bioelectric fields of the organism. This perturbed, oscillating system forms complex, organized ion-electron pulses which are associated with trans-membrane electric fields originating within each cell and within each tissue type. The envelope of each CDP trace follows a log-time function quite precisely; a relationship which is indicative of an unstable, dissipative system in which the current density exhibits a systematic decrease with time—resulting from a limited reservoir of reaction sites. The ubiquitous nature of continuous, low amplitude CDP pulses suggests that their maintenance depends on active transport mechanisms which direct

homeostasis during the interval of biofield perturbation (tissue contact). A maximum energy output (P_a) and the general form of each CDP trace is determined by complex resonance interactions between the individual biofields of contiguous types of tissue. In living carrot tissue the maximum CDP current amplitudes (P_a) were found in the tissues most active physiologically (phloem and primary xylem) and from linear regression analyses of log-time dissipation curves the Gibbs free energy of activation (E_a) in *Dacus carota* tissue was 9.4 kcal/mole. An E_a of 9.4 kcal/mole is somewhat lower than most chemical reactions; however, the energy barrier for activated CDP waves can be reduced by intercellular soliton formation, as for example in self organized electron-phonon associations. The discovery of a sharp reduction in the dissipation rate constant at 40°C is a clear indication of a close association between CDP formation and cellular metabolism.

At the tissue-electrode interface, complex charge carriers are formed and the observed non-ohmic conductivity patterns are suggestive of superconductivity type mechanisms in Josephson Junction systems.[12] The characteristics of the CDP waveforms are related to the energy band gap for electron conduction in the metal used for the electrode system. Hand exposure experiments indicate that photons in the UV region may induce reactions of the electron-phonon type.

In 1957, Albert Szent-Gyorgyi addressed the question "how does energy drive life?" He focused on the problem of how the high energy phosphate bond, P^*, in the ubiquitous Adenosine Triphosphate (ATP) molecule is utilized in living systems.[17] He started by making the distinction between *bond energies* and *excitation energies.* Bond energies are enclosed within molecules and have no outward action; this fact is represented by the symbol (E). Excitation energies are mobile and may interact with their surroundings; this fact is represented by the symbol E^*. So the problem "how does energy drive life?" may be expressed by asking a question of the form: "Is the (E) of ATP exchanged for E^* in the situation to be understood?" In the present context this means asking: "Can the generation of biofield energy, as detected by the CDP device, be understood as an exchange of the (E) of ATP for E^*? Later in his book Szent-Gyorgyi refers to the energy of P^* as "the biological energy unit," with a value of the order of (in modern notation) 10.0 kcal/mole.[17(p.24)] He goes on to point out that a photon of this energy has a wavelength of 2-3 micrometers, corresponding to the near infrared.

More recently Harold refers to "the proton-translocating ATPase" in the context of a discussion of the Chemiosmotic Theory of energy coupling by ion currents.[18] He points out that the proton-translocating ATPase is known to generate a proton-motive force on the order of 4.6 kcal/mole. Since the free energy of ATP hydrolysis in the cytoplasm is about 10.0 kcal/mole, the data are consistent with the transport of at least two protons per cycle. If we take Harold's quoted value of 4.6 kcal/mole for the proton-translocating ATPase the corresponding figure for two protons is 9.2 kcal/mole. The experimentally determined figure of 9.4 kcal/mole reported above for heated carrot root is within 2.2% of this figure. This result strongly suggests that the generation of biofield energy, as detected by the CDP device, might well be understood as an exchange of the (E) of ATP for E^* and, consequently, that the CDP device may have considerable potential as a tool for the study of Szent-Gyorgyi's problem "how does energy drive life?" by providing access to quantitative aspects of fundamental bioenergetic processes in intact living systems.

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ACKNOWLEDGMENTS: It is with deep sadness that I (WCL) report the death of my wonderful friend and associate Dr. John L. Gedye. His deep intellect, great dignity and charming sense of humor, provide us with a robust memory. Also we wish to acknowledge the steadfast support of our research by Dr. Dean Black and Mr. Frank Lucatelli.

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