A Methodological Issue in the Study of Correlation between Psychophysiological Variables

By

E. C. May, S. J. P. Spottiswoode, and L. V. Faith
Cognitive Sciences Laboratory
Laboratories for Fundamental Research
Palo Alto, California US

Abstract

We used previously accumulated skin conductance (SC) and EEG data to examine the effects of their respective autocorrelations upon hypothesis testing. We found that SC data remain autocorrelated for many seconds, and that EEG data remain autocorrelated for many fractions of a second depending upon filtering parameters. We show that the effect of these non-zero autocorrelations upon the interpretation of correlation coefficients using normal statistics can lead to substantial and artifactually inflated significance levels. With SC, for example, the high autocorrelation can lead to a Pearson's *r* correlation of 1.0 *even under the null hypothesis.* The resulting null-hypothesis z-score distribution range is [-20,20]; whereas, is should be approximately in the range [-3,3]. Alpha EEG, while less autocorrelated than SC, still leads to Pearson's *r* correlations in the range [-0.4,0.4] leading to a null-hypothesis z-score distribution in the range [-15,15]. Beta band EEG reduces the null-hypothesis z-score range to [-5,5]. We demonstrate that standard Monte Carlo techniques can provide valid estimates of the significance levels. The underlying assumptions of conventional statistical tests can be easily ignored, and the resulting error may become embedded into the thinking of a research community. As an example, we critically review a paper claiming significant correlation between the EEG's of isolated subjects (Grinberg-Zylberbaum, Delaflor, Attie, and Goswami, 1994); however, using uncorrelated EEG data from one of our previous studies and Monte Carlo methods to model the true null hypothesis, we compute a non-significant difference (Z = 1.22) between their non-"correlated" subjects and their "correlated" ones. As a result of their, possibly incorrect, interpretation of these correlations there is a growing literature proclaiming that these experiments are evidence for EPR-like quantum connections in isolated brains. These putative connections have been used as explanations, or at least plausibility arguments, for a variety of phenomena including distant healing.

Introduction

The field of research parapsychology has evolved to the point that there is now incontrovertible evidence in the free-response data of a statistical information transfer anomaly. In other words, we must reject the null hypothesis.[1] Some of the recent meta-analyses that address issues of methodology, replication, and summary statistics can be found in Utts (1991, 1996), Bem and Honorton (1994), and Radin (1997). Note that we have been careful not to claim that the existence for ESP has been proved. Rather, given that ESP has a negative definition—it is what happens when nothing else should—the declaration of an anomaly is the only valid statement that can be made.

One of many possible avenues toward trying to understand the underlying mechanisms of this anomaly (hence forth called ESP) is to search for psychophysiological correlates. There is a substantial literature describing many different approaches, but it is beyond the scope of this paper to delve into an analysis of this substantial work. Instead, we will focus on one technical aspect.

With the decrease of cost and increased capability of psychophysiological hardware and software it becomes increasingly attractive and feasible to monitor psychophysiology of isolated participants in a variety of circumstances in ESP experiments. One method, for example, would be to search for correlations between data epochs of isolated pairs of subjects.

Our paper reviews the well-known underlying assumptions on correlation statistics, shows examples from skin conductance and EEG data of what happens if the assumptions are violated, and suggests ways to use Monte Carlo techniques to obtain valid statistics when these assumptions are violated. In addition, we provide a critical analysis of one set of published experiments claiming an ESP-transferred event-related potential from one isolated subject to another (Grinberg-Zylberbaum, Delaflor, Attie, and Goswami, 1994).

Method of Approach

It is not the purpose of this paper to provide an exhaustive methodological critique of previous studies nor do we wish to provide a complete view into the complex methodologies associated with the measurement of psychophysiological variables. Rather, we will focus on a single issue: what is the correct approach to measure a correlation between psychophysiological variables?

We have all been taught that a clear understanding of all the explicit and implicit assumptions is absolutely necessary before applying statistical formulae to a problem. However, while we may believe this to be important, it is often overlooked when it comes time to actually analyze a study.

Let us review the familiar Pearson's correlation coefficient, r:

[1] At the 20th Congress of the Committee to Scientifically Investigate the Claims of the Paranormal (CSICOP) during a symposium of the existence of PSI, Utts said that the evidence is such that the null hypothesis must be rejected and that proponents and skeptics alike would be better served to expend their resources toward understanding the anomaly. In his "rebuttal" remarks, Hyman said, "I agree." One of us (May) was in the audience during that meeting.

$$r = \frac{\sum_{i=1}^{n}(x_i - \bar{x})(y_i - \bar{y})}{\sqrt{\sum_{i=1}^{n}(x_i - \bar{x})^2 \times \sum_{i=1}^{n}(y_i - \bar{y})^2}},$$

where *n* is the number of pairs of data points and *X* and *Y* are vectors containing the data. As we recall, the assumption is that both *X* and *Y* are both distributed normally and are *random* variables (i.e., the data points in *X* are independent of each other and the data points in *Y* are independent of each other).[2] The means of *X* and *Y* are *x-bar* and *y-bar*, respectively. If these assumptions are met, there are a number of methods to assess the significance level of a given *r* for *n-2* degrees of freedom. For example, we can use a Fischer's Z transform to arrive at a Z-Score that is distributed, under the null hypothesis as *N(0,1):*

$$Z = \sqrt{n-3} \times 0.5 \ln\left(\frac{1+r}{1-r}\right),$$

or a Student's t representation as:

$$t = r\sqrt{\frac{n-2}{1-r^2}}, \quad df = n-2.$$

We will only examine the degree to which the Z-score formalism holds for actual psychophysiological data, because the underlying assumptions for the T-score formalism are similar.[3]

Test Data Sets

To represent the central nervous system, we used 5-minutes of occipital (O1) EEG data that we collected from two different people on two different days. These data were part of our event-related desynchronization study that we carried out in 1996 at Stanford University (May et al., 2000). To represent the autonomic nervous system we used skin conductance DMILS data from a study at Edinburgh University.[4] We randomly selected five minutes of data each from two different subject who's sessions were on different days. Thus with both data sets we assume that the expected correlation is zero.

Analysis—Autonomic Nervous System Variables

As an example of an autonomic nervous system variable, we begin with the skin conductance (SC) data. It is well known that SC varies slowly and is not random. That is, each new data point contains a "memory" of the previous data points. Figure 1 shows the autocorrelation function for one of the SC data sets.

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[2] We can relax the normality requirement by using a non-parametric correlation such as Spearman's ?, however the random variable assumption remains.

[3] Often in EEG research coherence is used rather than a correlation measure. Coherence measures usually involve FFT's which are linear transforms of the data. Such a transform will not remove the autocorrelation so the interpretation problems will remain. We will not, therefore, include a coherence analysis in this paper.

[4] We thank Professor Deborah Delanoy for granting us access to her data from an as of yet unpublished sender/no sender DMILS study.

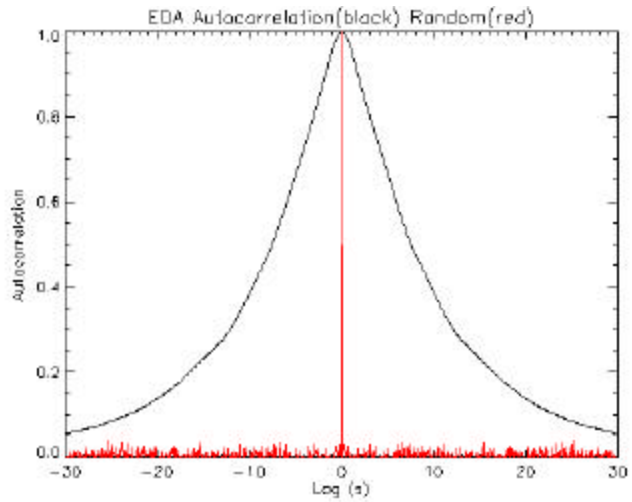

Figure 1. Autocorrelation of Skin Conductance and Random Data

We compute the autocorrelation for lags from -30 to +30 seconds. The noisy curve with a "spike" at zero lag is the autocorrelation of normally distributed random data. We notice that there is a correlation of 0.5 for lags as long as about eight seconds. That means that the SC data point being measured now contains information about the SC data eight seconds previously.

Clearly this is a strong violation of the random-variable assumption built into both the Pearson's *r* and Spearman's *?* correlation functions. The critical question is to what degree does a strong skin conductance autocorrelation affect a statistical evaluation?

To address this question, we used a Monte Carlo technique. For each of 1,000 passes, we randomly selected 10-second intervals (i.e., 160 points), one each from the two independent SC data records. In addition, for each pass, we computed two separate random vectors of the same length as the SC data, which were distributed as *N(0,1)*. We computed a Pearson's *r* and a Spearman's *?* for the pair of SC data and for the random data. The distributions are shown in Figure 2.

The effect of having such a strong autocorrelation is immediately obvious in Figure 2a. The correlation of random data, which is shown as the light (green) Gaussian-shaped histogram centered on zero, ranges from about –0.25 to +0.25 as expected for correlation coefficients with 158 degrees of freedom. The surprise is the nearly uniform distribution of correlation coefficients for both Pearson's *r* (darkest) and Spearman's *?*, which are shown as the more uniform darker distributions (blue and red, respectively) The peaks of the distributions near unity correlation is expected for non-specific skin conductance in that the two subjects were essentially relaxing in the absence of overt stimuli. So both SC records tended to decrease throughout the 10-second epoch leading to an excess of large positive and negative correlation values.

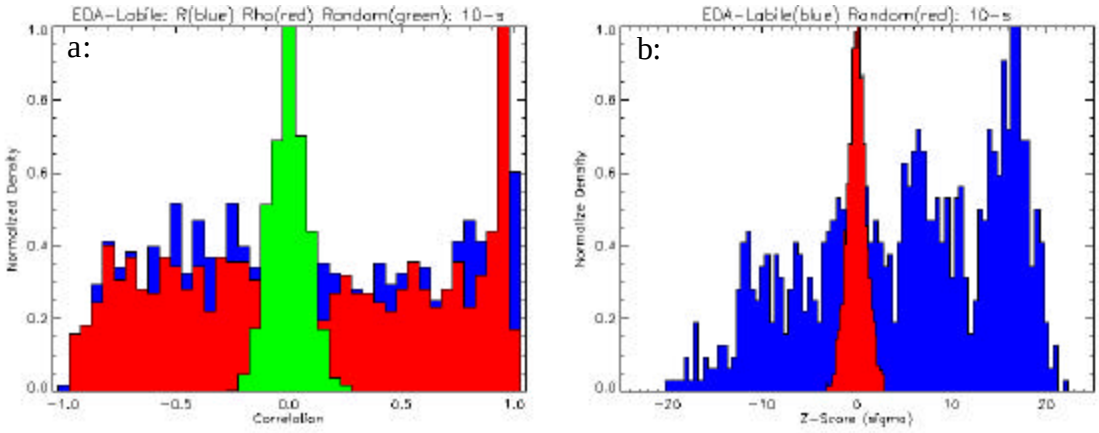

Figure 2. Skin Conductance: Correlation and Random Data.

Figure 2b shows the distribution of Z-scores computed from the Fischer's Z as shown above. The lighter (red) histogram centered on zero shows the expected range of Z-scores for uncorrelated random data that meet all the criteria for the proper interpretation of Pearson's *r*. The highly autocorrelated SC data on the other hand, which fails the random-variable criterion, and is shown as the darker (blue) histogram in Figure 2b shows Z-scores that range from -20 to +20 even for totally uncorrelated data.

The proper way to asses a probability of an observed correlation between epochs which themselves are highly autocorrelated is to use a Monte Carlo technique.[5]

To demonstrate how this works, consider the darkest (blue) histogram shown in Figure 2a above. We generated that histogram from the correlation of 1,000 pairs of 10-second SC epochs. Suppose we wish to compute a Z-score for one of those observed correlations, r_o. The following are the steps to compute a valid 1-tailed Z-score:

1. Order the 1,000 element correlation vector from smallest to largest.
2. Compute how many correlation values are greater than or equal to r_o.
3. Break ties by computing the center position among the ties.

The p-value is computed as:

$$p = \frac{number of\ r's \geq r_o}{total number of\ correlations}$$

This p-value can be converted into a Z-score by the usual method. Figure 3 shows the distribution of 10,000 Z-scores resulting form the Monte Carlo calculation described above.

[5] We do not claim to be the first to come up with the idea of applying Monte Carlo techniques to assess probability. This technique can be found in the literature under such names including permutation technique—partial or complete and bootstrap methods.

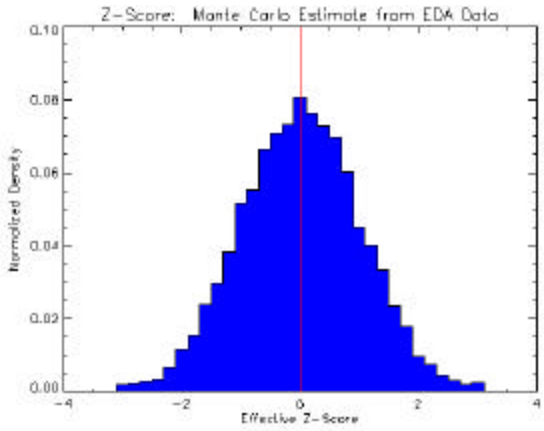

Figure 3. Z-Score Distribution from Monte Carlo Calculation

This is a well-behaved *N(0,1)* distribution that is expected under the null hypothesis of no correlation even in the case of highly autocorrelated data. Although the standard formalism to compute Z-scores from correlations must be rejected in autocorrelated data, a Monte Carlo calculation can give reasonable non-assumptive and non-parametric estimates of the likelihood of such correlations given the data sets at hand.

We have used skin conductance to illustrate the problems associated with a slowly varying autonomic nervous system variable; however, the same problems will exist with other such variables that include heart rate, respiration rate, and blood volume. We recommend the use of Monte Carlo methods to make statistical assessments for all correlations among these types of variables.

Analysis—Central Nervous System Variables

The problem of correctly assessing correlations also exists with the central nervous system data, though less severely. We filtered 5 minutes of occipital (O1) EEG data from 8 to 10 Hz and computed the auto correlation for lag between -1 and +1 second. For comparison, we generated the equivalent length of random data which was distributed as *N(0,1)*.[6] Both autocorrelation functions are shown in Figure 4. The "spike" at zero lag is for the random data as expected. In the alpha EEG case, the autocorrelation function, while significantly shorter than for skin conductance data, nonetheless is significantly longer than for random data. In Figure 4, we have shown both plus and minus lags to be complete; however, only the negative lags have meaning. That is, EEG alpha "memory" lasts for approximately one half second, or the EEG alpha value being measure "now" contains information about the EEG alpha value as much as 0.5 seconds earlier.

[6] For psychophysiological data, there are potentially two sources that contribute to the autocorrelation function. The first is inherent in the system, and the second results from applying filters to the data. In this paper, we show random data which is distributed as *N(0,1)* to illustrate the effects of non-zero autocorrelations.

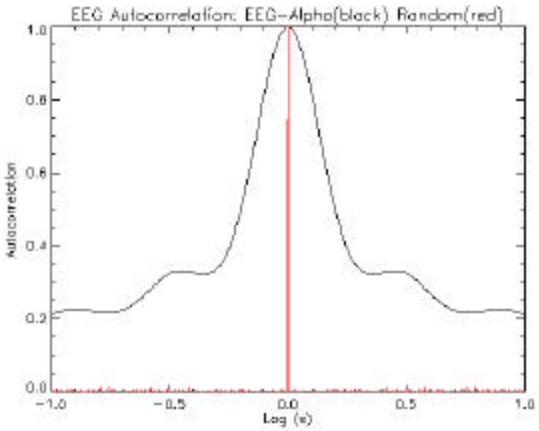

Figure 4. Autocorrelation of EEG Alpha and Random Data

The question is to what degree does this shorter autocorrelation influence the statistical assessment of correlations between two such sets of data. We randomly selected 1,000 10-second epochs from each of the two EEG records and computed correlations (i.e., Pearson's *r*) for each epoch pair. Figure 5a shows the distributions of these correlations.

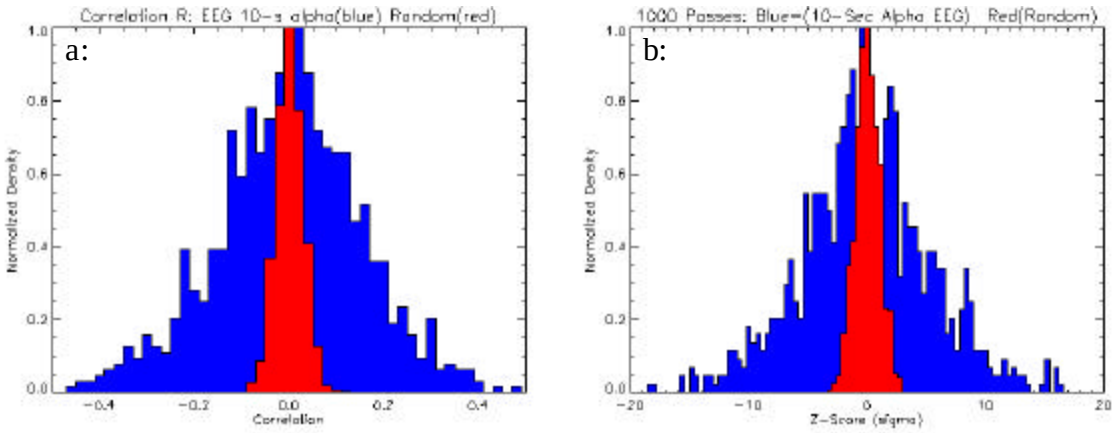

Figure 5. Correlation Distributions of EEG-a and Random Data.

The wide, darker (blue), nearly Gaussian shape is the distribution for the alpha, whereas the narrow, lighter (red) distribution is for random data. So even with a sharply reduced autocorrelation function compared to skin conductance, nonetheless, EEG-a correlations can be seriously misleading. Figure 5b shows the equivalent Z-scores distributions.

We notice that the Z-score distribution for the random data is as expected; however, as in the skin conductance case, the Z-score distribution for the EEG data can lead to wildly incorrect answers.

As in the case of skin conductance data (please see Figure 3), a Monte Carlo approach gives a *N(0,1)* distribution and allows for a valid assessment for correlation between separate central nervous system alpha epochs.

Analysis Summary

We have shown that the underlying assumptions that would allow the use of a Fischer's Z transform to compute the significance levels of a Pearson's *r* correlation are substantially violated with autonomic and central nervous system data. Of course this is a well known result; however, in the next section we will show an example of a "land mark" paper that ignored these difficulties.

Discussion

Grinberg-Zylberbaum (1982) became interested in studying EEG correlation between communicating individuals, and he and his colleagues have been publishing similar studies for over a decade.[7]

The basic idea behind this research is to examine correlations between individuals at various stages of communication. For example, subjects were sequestered together in the same room (Grinberg-Zylberbaum & Ramos, 1987) and broad-band (i.e., 3 - 45Hz) EEG was measured simultaneously, or in isolated chambers using a "telepathic" paradigm (Grinberg-Zylberbaum, Delaflor, Arellano, Guevara & Perez, 1992). Pearson's *r*'s were computed and displayed graphically for visual inspection and quantitative analyses. Grinberg-Zylberbaum and Ramos (1987) observed what they described as strong coherence between these sets of EEG records and presented means and standard errors for the correlations under various conditions. Since they do not mention any Monte Carlo methods, we assume they used normal statistics

For the remainder of this discussion, we will focus upon a paper by Grinberg-Zylberbaum, Delaflor, Attie, and Goswami (1994) because it is the most detailed with regard to methodological issues and is often referenced as the quintessential example of quantum coherence between isolated brains.[8] Hence forth we will refer to this paper as ZDAG.

ZDAG Overview

The basic idea behind this paper was to observe event related potentials (ERP) in the EEG record of an isolated individual while a second isolated individual was being stimulated with 100 random light flashes—the details of which were not described in the paper. EEG was also monitored from the subject who was directly stimulated. There were two conditions of interest:

1. Before Interaction. In this condition called Condition 1 in the paper, the above measurements were made between individuals who had not met or interacted in any way.
2. After Interaction. In this condition called Condition 2 in the paper, the individuals were introduced to each other inside the stimulation chamber and instructed to "get to know" each other and then to "feel one another in meditative silence" for 20 minutes before the above EEG measures were obtained.

[7] It is not our purpose to single out this group for our critical remarks; however, their pioneering research has invoked substantial experimental, theoretical, and philosophical interest.

[8] We will only focus upon the methodologies associated with the correlation calculations and the resulting conclusions rather than providing a complete critique of the whole paper.

Standard ensemble averaging was performed over the 100 stimuli for the "sender" from zero to about 0.5 seconds relative to the stimulus, and using the same stimuli markers, a similar ensemble average was carried out for the "receiver's" EEG record.

All EEG records were filtered between 12.7 and 35 Hz prior to the ensemble averaging. The goal was to observe an ERP in the sender and see a correlated ERP in the receiver. For 48 steps of 16 samples each, a Pearson's r was computed. Thus, a temporal record of the correlation could be observed for about 0.5 a second relative to the stimulus. The authors do not indicate how significance levels were computed, a quantitative statistical measure between Conditions 1 and 2, nor do they mention whether any Monte Carlo methods were used; thus we presume they used a "standard" *Z* or *T* method.

Within the first 132 ms, they report correlation levels ranging from 0.700 to 0.929 corresponding to $p < 0.009$.[9]

Independent Evaluation of Mean Chance Expectation

For our evaluation, we used two 5-minute EEG records from different days and different subjects from our 1994 ERD study. Our assumption is that there is no real correlation between these isolated records.

Following the procedure described in ZDAG, we filtered both EEG records from 12.7 to 35 Hz and arbitrarily labeled one of the EEG records as the "sender" (i.e., the subject who experienced direct stimulation) and the second as the "receiver." For each of a 2,500 pass simulation, we:

- Randomly selected 100 entry points into the sender's record to simulate 100 stimuli.
- Identified the same entry points into the receiver's record.
- Ensemble averaged 64 data points (0.5 seconds) starting at each of all 100 stimuli separately for the sender and receiver EEG records.
- Followed ZDAG's computation and computed four 16-point Pearson's *r* correlation coefficients between the sender and receiver ensemble averaged data in each 64-point epoch.

Thus we computed a total of 10,000 values for the correlation coefficient. At the same time we computed 10,000 correlation coefficients for random variables, which were distributed as *N(0,1)*. Figures 6a and 6b show these distributions and their equivalent Z-score distributions under the assumption of normal statistics, respectively.

[9] It is not clear how this value was computed since it does not correspond to any correlation in the range quoted in the paper.

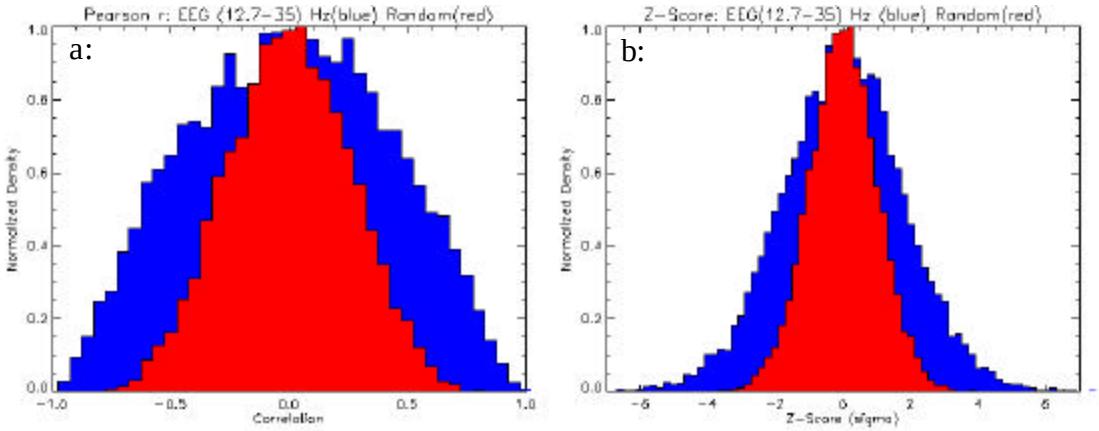

Figure 6. Pearson's r for ERP Simulation and Random Data

The lighter (red) distributions are associated with the random data and, and the wider darker (blue) distributions are associated with the correlations as described above. As we can see, even for this relatively high frequency EEG (i.e., 12.7 to 35 Hz), their remains considerable difference between truly random data and uncorrelated EEG.[10]

We computed a Z-score distribution resulting from a Monte Carlo calculation, similar to that described above so that we could compute a valid Z-score for a given observed Pearson's *r*.

The consequences for the differences between the EEG correlation and that for random data can be seen in Figure 7. Note that the p-value is 1-tailed and is shown on a log scale.

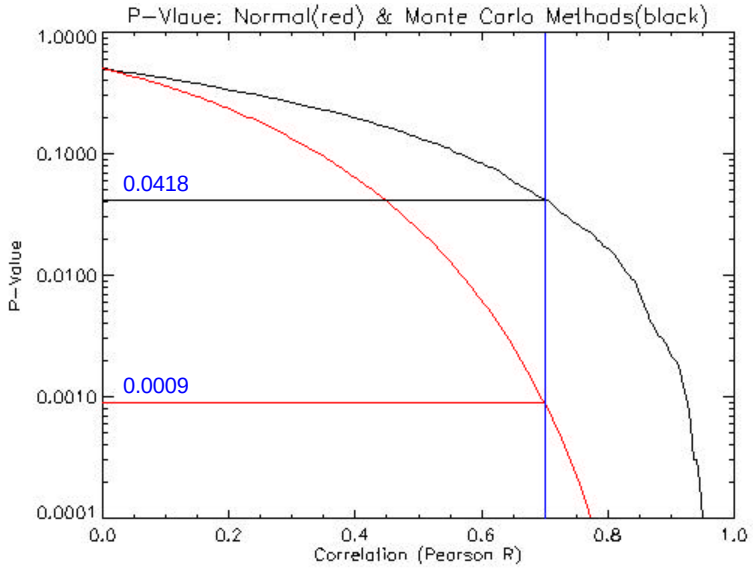

Figure 7. P-Values Under a Normal and Monte Carlo Assumption.

We have chosen to illustrate an artifactual enhancement of the p-value for a correlation value of 0.7 because that was the minimum value quoted in ZDAG. At larger values of

[10] The effects of frequency can be seen by comparing Figures 5b and 6b. The lower frequency gives a much broader Z-score distribution for mean chance expectation than does the higher frequency EEG.

the correlation the artifact becomes even larger. As Figure 7 shows, a p-value of 0.0009 will be found using normal statistics; whereas the correct value is 0.0418.

Unfortunately no correlation values were given for Condition 1 (i.e., before the sender and receiver met); however, selected graphical representations for the correlations are shown for Condition 1. ZDAG did not compute any quantitative difference between their conditions. If we assume, however, as ZDAG implies that Condition 1 yields no correlation (i.e., Z-score = 0) then there is no significant difference between the conditions for an observed correlation of 0.7:

$$Z_{diff} = \frac{(1.73 - 0.0)}{\sqrt{2}} = 1.22,$$

where the value 1.73 is the Z-score equivalent of the one-tailed p-value = 0.0418 shown in Figure 7.

The selected graphical representations for Condition 2 appear quite impressive until one realizes from Figure 7 that there is a sizeable likelihood of large correlation value even under the null hypothesis. So with selected epochs it is not surprising to see rather impressive overlaps between the sender and receiver ensemble averaged data.

We computed the autocorrelation function for this set of EEG data and found that although it was narrower than it was for alpha, nonetheless, it is responsible for the breaking of the underlying assumption for the use of normal statistic and the resulting invalid p-values.

Unfortunately, there is insufficient reporting in ZDAG to determine without question whether their claim of isolated EEG evoked responses are correlated is correct; however, in light of our results we urge caution in interpreting this and similar papers.[11]

Commentary

As we have indicated above, there have been a number of references to Grinberg-Zylberbaum and his colleague's work. In this section we provide quotes from three of these articles selected from a list of papers in the Bibliography Section below. The purpose of this section is to demonstrate how a concept can become embedded into the research community even though it may be in error.

For example:

> Dossey, L. (1997). "Grinberg-Zylberbaum's team, along with physicist Amit Goswami, propose that these 'transferred potentials' between brains demonstrate 'brain-to-brain nonlocal EEG correlations.' Nonlocal correlations have been a concern of physicists since they were proposed by Einstein, Rosen, and Podolsky in 1935. From the moment they were hypothesized, nonlocal effects have stretched the imagination of physicists to the limits. The fact that they occur simultaneously between distant subatomic particles means that there is no "travel time" for any known form of energy to flow between them. But if there is no signal from one particle to the other, how could their behavior be correlated? How could one particle know what the other is up to? For almost half a century, nonlocal events

[11] In ZDAG, the authors claim an EPR-type correlation, yet do not address such critical issues of the quantum correlation that is required (i.e., their Condition 2) in the light of substantial evidence that environmental decoherence sets in at nanodegrees Kelvin; whereas the brain is at 300^0 Kelvin.

remained hypothetical--until they were demonstrated experimentally, most notably in a celebrated study in 1982 by physicist Alain Aspect and colleagues.

Physicists have assumed that nonlocal connections exist only between subatomic particles such as electrons and photons. But the pioneering work of Grinberg-Zylberbaum, Goswami, and colleagues strongly suggests that these events occur also between human beings."

Andrews, S (1996). "In fact, several generations of scientists have amassed evidence in support of remote communication anomalies. Numerous studies have provided evidence that identifiable and consistent electrical brain signals (as distinguished from electrical brain signals occurring during control periods) occurred in one person when a distant second person was either meditating or provided with sensory stimulation, or when a distant person attempted to communicate with the subject being monitored."

Goswami, A. (1999). "The striking similarity between the correlated brains of this experiment [Grinberg-Zylberbaum, Delaflor, Attie, and Goswami, 1994] and the correlated photons of Aspect's should be clear, but there is also a striking difference. The similarity is that in both cases the initial correlation is produced by some 'interaction.' In the case of the photons, the interaction is purely physical. But in the case of the correlated brains, consciousness is involved. For correlated photons, as soon as the possibility wave of one is collapsed by measurement, the objects become uncorrelated. But in the case of the correlated brains, consciousness not only establishes correlation initially but also maintains the correlation over the duration of the experiment. To get a clear evoked potential, experimenters typically use an averaging procedure over one-hundred or so light flashes in order to eliminate the 'noise.' But the brains do not become uncorrelated as soon as one observer sees a light flash. The only conclusion is that consciousness reestablishes the correlation every time it is broken.

This difference between correlated photons (as in Aspect's experiment) and correlated brains (as in Grinberg-Zylberbaum's experiment) is highly significant. The nonlocality of correlated photons, although striking in terms of demonstrating the radicalness (*sic*) of quantum physics, cannot be used to transfer information, according Eberhard's theorem. Each photon that one experimenter sees in a stream of photons is correlated with its partner that is observed by another experimenter. But there is no correlation between the states of the photons within the stream observed by one experimenter. These states thus are randomly distributed, hence they can carry no message. But in the case of the correlated brains, since consciousness is involved in establishing and maintaining the correlation over the period of the entire experiment, Eberhard's theorem does not apply, and message transfer is not forbidden."

Conclusion

We have demonstrated that substantial errors can be made if the underlying assumptions concerning hypothesis testing with correlations are violated, and these errors can propagate with the research community. We used skin conductance and EEG data from previous experiments to show that even small autocorrelations can affect the resulting p-values by up to many orders of magnitude.

Fortunately, we were also able to demonstrate that by using standard Monte Carlo techniques, is its possible to extract meaningful hypotheses test statistics.

Given the simplicity of encoding Monte Carlo analyses into Microsoft's Excel spread sheets and other programming languages, we urge our colleagues to adopt this method in all further psychophysiological correlation studies.

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