Dead Science in Live Psychology: A Case Study from Heart Rate Variability (HRV)

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Abstract

Psychological theories often build from theoretically separate fields in the biosciences – physiology, biology, neuroscience, etc. – to situate human behavior within the body. However, these are increasingly sophisticated areas of research which rapidly change and adapt their evidence base. The current paper is a case study examining what happens to psychological research when its foundational biological context is invalidated or superseded. The example we use is heart rate variability (HRV) as a purported measure of cardiac sympathetic outflow. While objections to this technique within physiological research have been established and confirmed for decades, its false status continues to be maintained in applied psychological research. We review a combination of factors within scientific and publishing practice, practical and conceptual barriers to experimental interface, and personal/professional value of the invalidated theory in attempt to understand how dead science can be kept alive in psychological science.

Keywords: LF/HF, heart rate variability, autonomic nervous system

Dead Science in Live Psychology: A Case Study from Heart Rate Variability (HRV)

Heart rate variability (HRV) refers to beat-to-beat changes in the duration of the electrocardiographic R-R interval, which corresponds to intervals between ventricular depolarisations of the heart. HRV is popular in psychological science because it allows for low-cost, non-invasive, and accessible autonomic information. Beginning from initial observations of cardiovascular and circulatory rhythmicity in the 19th Century (Billman, 2011), there are presently more than 1,000 papers employing or assessing HRV published each calendar year, the continuation of a trend beginning in the early 1990s.

Contrary to the ways many psychological researchers employ HRV, the modulation of heart rate (HR) over time is complex. It is influenced variously by autonomic, cardiac, respiratory, circulatory/vascular, hormonal, neuropeptidergic, etc. factors. Nevertheless, strong control of the respiratory cycle over beat-to-beat changes and its relatively unambiguous control via the efferent vagus mean HRV is broadly interpreted as reflecting fluctuations in cardiac parasympathetic tone (Eckberg, 1983). To quantify these fluctuations, HRV is most commonly assessed within the frequency domain; specifically, a beat-to-beat HR series is resampled into equally spaced intervals, detrended, and power spectral density of frequency bands corresponding to physiological processes are calculated by Fast Fourier Transform or similar. These HRV values can be compared between or within subjects.

As above, respiratory influence over instantaneous heart beat via the cardiac vagus determines that cardiac parasympathetic outflow can be approximated by the power spectral density at “high frequency,” i.e., the speed of breathing (0.15-0.4Hz). A common interpretation of the lower frequency band (0.04-0.15Hz) has two central hypotheses, which are:

Hypothesis A: Cardiac sympathetic outflow can be approximated by the power spectral density of the 0.04-0.15Hz band; hence,

Hypothesis B: The ratio between frequency bands (where 0.04-0.15Hz is proportional to cardiac sympathetic outflow as above, and 0.15-0.40Hz is strongly determined by cardiac parasympathetic outflow) can form an index of 'sympathovagal balance.'

Accordingly, Hypothesis B is necessarily reliant on Hypothesis A. As this 'balance' can be deployed as a simple global measurement of overall arousal, autonomic readiness, or health status, it has been widely used as a dependent variable in studies of emotion, panic, stress, attentional state, health status, and so on in psychological science.

The relevant theory behind measures of sympathovagal balance has been outlined in detail (Baselli et al., 1986; Lombardi et al., 1987; Malliani, Pagani, Lombardi, & Cerutti, 1991; Pagani et al., 1984, 1985, 1986). The central papers within this body of work (Malliani et al., 1991; Pagani et al., 1986) have 3,608 citations and 4,134 citations respectively to date (Google Scholar; 08/2017). While the limitation of available search tools prevents an authoritative answer to how commonly these hypotheses are deployed in published research, a recent estimate found 26 out of 97 papers (~25%) using any form of frequency-based analysis offered a direct or qualified statement of Hypothesis A, and 74 out of 97 (~75%) reported at least one ratio measure, i.e., offered a calculation reliant on Hypothesis B (Heathers, 2014). In 2016, a minimum of 166 papers were published making explicit mention of Hypothesis B¹. Both hypotheses are cited broadly within

¹ [PubMed search](#); LF/HF or normalized units

applied physiology (e.g., exercise and medical science), and journals with a specific focus on the physiological underpinnings of psychological processes are no exception (Figure 1).

Figure 1: Journal references to two common instantiations of Hypotheses A and B; journals included are: *International Journal of Psychophysiology*, *Psychophysiology*, *Biological Psychology*, *Applied Psychophysiology and Biofeedback*, *Biological Psychiatry*, *Journal of Psychophysiology*.

However, contemporary autonomic and circulatory physiology widely regards these hypotheses as problematic. Table 1 collates the conclusions of 20 relevant review papers that address the two hypotheses of interest both directly and obliquely, and in various amounts of detail. They variously consider the: (a) conceptual and methodological basis of the hypotheses; for instance, the possibility of measuring sympathetic outflow from HRV considering the kinetics of

Sympathetic Nervous System (SNS) action at the AV node; (b) experimental environment used to initially determine the hypotheses (i.e., graded orthostasis); (c) relationship between the hypotheses and relevant conditions of altered cardiac sympathetic outflow (heart failure, exercise, etc.); (d) relationship between the hypotheses and similar techniques for assessing sympathetic outflow; and (e) nature and role of the circulatory system in Hypothesis A.

Reference	Text
Berntson et al., 1997	"In the absence of explicit validation over a broad range of conditions ... the LF/HF ratio cannot be considered as a specific index of sympathetic cardiac control or sympathovagal balance".
Eckberg, 1997	"This review calls attention to major problems with the construct of sympathovagal balance ... sympathovagal balance may obscure rather than illuminate human physiology."
Grassi & Esler, 1999	"Low-frequency variability in heart rate does not, of course, strictly provide a measure of the rate of firing of the cardiac sympathetic nerves, despite widespread and often uncritical interpretation of the variability measurement in this way."
Eckberg, 2000	"There is, to my knowledge, no evidence showing that low-frequency R-R interval spectral power in resting humans bears any quantitative relation to sympathetic neural outflow."
Esler, 2000	"Although the low-frequency heart rate variability (approximately 0.1 Hz) derives in part from the influence of the cardiac sympathetic nerves, it is very misleading when used as a measure of cardiac sympathetic firing."
Notarius & Floras, 2001	"...Frequency domain measures of heart rate variability, obtained under resting conditions, should not be used to derive inferences as to differences in central or cardiac sympathetic outflow in individual heart failure subjects."
Malpas, 2002	"One approach with regard to heart rate variability has been to calculate the ratio of the two spectral powers in each frequency band as an index of sympathovagal balance ... A number of major criticisms have been raised that question the validity of such an index."
De Jong & Randall, 2005	"Some investigators argue that the ratio of power within the low frequency vs. high frequency spectral regions ... distinguishes sympathetic effects from parasympathetic effects. However, this is controversial and caution is warranted in drawing any conclusions in this regard."
Kleiger, Stein, & Bigger, 2005	"Various manipulations of high and LF power, e.g., normalization or the LF/HF ratio has been applied in an attempt to better estimate sympathetic activity. These manipulations are based on a somewhat simplistic "ying-yang" model of cardiac autonomic function. Results have been illuminating under some circumstances (e.g., tilt table testing) and readily misinterpreted under others (numerous papers in which increases in the LF/HF ratio due to reductions in HF power have been interpreted as increased sympathetic activity)."

Reference	Text
Freeman, 2006	"Low frequency R-R interval spectral power has been proposed as a pure index of sympathetic activity, however, recent evidence has not supported that notion."
Elghozi & Julien, 2007	"It is unwise to consider the amplitude of the 0.1-Hz HR oscillation as a surrogate measure of sympathetic nerve firing."
Goldstein et al., 2011	"Several lines of evidence, however, argue against the validity of LF power, with or without adjustment for HF or total power, as an index of sympathetic outflow especially to the heart."
Billman, 2011	"... The hypothesis that LF/HF quantifies "sympatho-vagal balance" depends upon four interrelated assumptions, all of which can be proven to be false. The facts are in direct opposition to the assumptions."
Papaioannou, Pneumatikos, & Maglaveras, 2013	"HRV analysis does not simply reflect sympathetic/parasympathetic balance ... any change in LF/HF ratio may correspond to central, baroreflex or cellular membrane effects of different stimuli during severe stress."
Reyes del Paso, Langewitz, Mulder, van Roon, & Duschek, 2013	"Evidence presented in this article challenges the suitability of the LF component of HRV as an index of cardiac sympathetic control and the LF/HF ratio as an index of sympathovagal balance."
Heathers, 2014	"the strong relationship between the baroreflex and LF power ... [is] extrapolated to the position that LF power is proportional to cardiac sympathetic nerve activity. The direct evidence against this claim is strong even if confined to just non-invasive or minimally invasive studies..."
McCraty & Shaffer, 2015	"it has been suggested that the LF band reflects sympathetic activity and the LF/HF ratio has been controversially used to assess the balance between sympathetic and parasympathetic activity. ... A number of researchers have challenged this perspective and have persuasively argued that in resting conditions, the LF band reflects baroreflex activity and not cardiac sympathetic innervation."
Draghici & Taylor, 2016	"The conclusions derived from HRV when using normalized units and/or LF/HF ratio should be carefully reconsidered."
Kiyono, Hayano, Watanabe, & Yamamoto, 2017	"...The clinical significance of the association of cardiac sympathetic overactivity with LF power-related indices is questionable."
Laborde, Mosley, & Thayer, 2017	"There is now a consensus to say that the precise physiological underpinning [of LF/HF] is unclear, thus lowering its predictive value ... we strongly recommend researchers to adopt HRV indices that reflect clearly identified physiological systems."

Table 1: A meta-review of statements considering a) the utility of LF HRV as an index of sympathetic cardiac outflow and b) the utility of LF/HF HRV as an index of sympathovagal balance. Order is year/alphabetical.

These 20 papers, aggregated over a period of 20 years, cite more than 60 primary sources, and universally conclude that these hypotheses are not well supported. While all the authors within Table 1 state the broad incompatibility of either hypothesis with the bulk of available physiological evidence, their recommendations for action range from noted caution through to active rejection. Readers with an interest in the status of Hypothesis A will find it directly addressed in Goldstein, Benthó, Park, & Sharabi (2011), and a comprehensive overview of the internal consistency and physiology underlying Hypothesis B in Billman (2013) and Karemaker (2017) respectively.

Given the above, how might such hypotheses persist within applied research? How might they maintain two separate and different scientific acceptabilities? The answer is complex, involving: (a) the theoretical distance between scientific fields; (b) the practical utility of the ideas (i.e., useful for the act of producing research, rather than producing knowledge); and (c) the structure and function of the scientific publication system itself.

Maintenance Factors

In the following we address eight potential factors which may singly or in combination allow the above outlined tension between physiology and the psychological sciences to be maintained. They include technical divergence, practical utility, conceptual utility, experimental success, explicit authority, implicit authority, dilution, and personal investment.

Technical Divergence a.k.a. 'Siloing'

Consider the following from a hypothetical academic introduction:

The sympathetic and parasympathetic nervous systems (PNS) have discrete projections to the heart; the SNS has several projections to the whole myocardium; the PNS exclusively

to the sino-atrial node (SAN). The SAN is a discrete area of tissue over the right atrium which acts as the 'pacemaker' of the heart. On a measured electrocardiogram, the initiation of the electrocardiographic cycle manifests as the P-wave, a small, positive, and regular wave corresponding to depolarisation/contraction of the atria. After propagation through the atrioventricular node (corresponding to the PR segment), the large typical 'spike' of the ECG is seen with depolarisation of the ventricles (the R-wave). The distance between R-waves, chosen because of their size and functional capacity (i.e., they are the direct precedent to the ventricular contraction which is the functional instantiation of the 'heart beat'), is considered the best representation of the cardiac cycle. Thus, the admixture of excitatory SNS and inhibitory PNS influences on the SAN determine the cardiac cycle.

The above is reasonably typical of an introduction to basic cardiac autonomic physiology, which readers of papers within biological psychology or psychophysiology would find reasonably familiar, if perhaps more detailed than usual. It is a reasonably standard paragraph-length outline of the relevant physiology for an applied audience, and well reflected in introductory textbooks on cardiac physiology and electrophysiology [e.g., Hall & Guyton (2011), p.123]. However, the above does not refer to a static body of knowledge, and almost the entire paragraph above can be heavily qualified or contradicted by the most recent available evidence:

- PNS and SNS influences on the heart are not discrete, they interact at both pre- and post-junctional levels; likewise, PNS and SNS influences on the myocardium do not represent monolithic, unipolar forces which always act in opposition (Coote, 2013).
- The PNS has projections not just to the sinoatrial node (SAN) but to the whole myocardium (Kawano, Okada, & Yano, 2003).

- The SAN is best described as an area rather than a discrete point (Boyett, 2009; Chandler et al., 2011).
- The myocardium is not a simple pump run by the admixture of efferent parasympathetic and sympathetic signals, rather it is a system with the ability to integrate information via the intrinsic cardiac nervous system, a network of ganglia and nerves on the epicardium (Armour, 2007; Armour, 2008).
- The P-wave is a complicated signal with both (a) characteristic changes between participants and (b) a non-stereotypical pathway whose stereotypical electrocardiographic signal is more a consequence of filter settings than its underlying physiology (Peper, Jonges, Losekoot, & Grimbergen, 1985; Potse et al., 2016).
- The distance between R-waves are overwhelmingly the most common representation of the cardiac cycle, but as the cycle begins with the P-wave, observed changes in the PR-interval (Carruthers, McCall, Cordell, & Wu, 1987; Shouldice, Heneghan, & Nolan, 2002) may obscure the true cycle length.

The point here is not that more recent conceptions of relevant cardiac and autonomic physiology invalidate all measurements of HRV. Rather, we submit that it is notable that every single foundational assumption listed in our 'typical introduction' has been overwritten. The above body of new knowledge is not arcane, but well established and drawn from active research programs primarily within cellular and cardiac physiology – it can be found in curricula beginning at around an advanced undergraduate level. However, this complete reconceptualization of the entire underpinnings of a measurement technique has only the most occasional bearing on the work which falls under the rubric of *applied* physiology, and a good demonstration that even well-connected fields of academic endeavor maintain a parochialism.

As HRV is a tool derived from an earlier body of knowledge, it retains its underlying assumptions in perpetuity even while the background it rests on is qualified or invalidated. Thus, we may see a similar attitude towards the research cited within Table 1 – these details, as central as they may be to contemporary physiology, might be regarded as extraneous to those using HRV as a correlate of autonomic status relevant to behavior. Consequently, physiological knowledge directly relevant to the work of social scientists who deploy physiological methods is 'siloes,' confined to a separate and unread body of literature. This may occur, as it does in this case, even if that evidence is extensive.

Practical Utility i.e. Ease of Access

There are several ways in which various instantiations of (SNS) outflow can be assessed. Measuring directly from the cardiac nerves is a destructive technique not possible in humans, and has only been attempted in large-animal models (Jardine et al., 2002). Within minimally invasive methods possible to use in humans, there are technical and hardware limitations; the measurement of plasma noradrenaline kinetics and noradrenaline spillover (Murray Esler, Jennings, & Lambert, 1989; Hasking et al., 1986) requires the use of injected radiotracers during a PET scan, and measuring muscle or skin sympathetic nervous activity directly requires specialized equipment, and the insertion of a microelectrode beneath the skin (Macefield & Wallin, 1996). None of the aforementioned specialist methods can be maintained in normal psychological laboratory conditions, and certainly not without modifying participant behavior and/or participation in typical behavioral experiments.

Non-invasive methods also exist, such as the measurement of salivary alpha-amylase (Rohleder & Nater, 2009) and quantification of sweat pore reactivity (Familoni et al., 2016), but

neither is widely established at present. The best established non-invasive method is the measurement of skin resistance/conductance (Boucsein, 2012), which has a long historical precedence (e.g. Hermann & Luchsinger, 1878), but can be difficult to interpret, displays a surprisingly complex sub-structure which is not well understood (Bach, Daunizeau, Kuelzow, Friston, & Dolan, 2011; Bach, Friston, & Dolan, 2013; Bach & Friston, 2013), and has an unresolved relationship with the directly-measured firing of skin sympathetic nerves (Bach, 2014; Henderson et al., 2012).

In contrast to the above, HRV measurement lacks barriers to collection – measurement is possible during movement and activities of daily living, is easily capable of taking multiple sequential measurements without participant fatigue, and is suitable for long-term recordings. It is also inexpensive, due to multiple commercially available hardware platforms and free software analysis programs. Finally, as per the original demonstrations, the method of using LF/HF ratio to index sympathetic outflow claims to be accurate – a seminal demonstration by Montano et al. (1994) showed correlations from 0.68 to 0.78 between the various instantiations of Hypothesis B and orthostatic tilt angle (an intervention where a lying experimental participant is gradually tilted upright on a table, where tilt angle is itself a well-constrained correlate of sympathetic outflow).

While objections to the meaning of this demonstration are long established (Berntson et al., 1997; Eckberg, 1997), the accuracy of the measurements themselves are not in dispute, and have been replicated (Porta et al., 2001). In other words, if we take the underlying theory as real, then Hypotheses A & B become attractive research tools to the psychologist interested in measuring sympathetic outflow, as would any experimental model presented as simultaneously cheap, convenient, accurate, and computationally straightforward.

Conceptual Utility

Researchers are faced with ever-increasing analytical and theoretical complications as the number of available scientific publications grows alongside the complexity of scientific knowledge and investigative tools. This multiply affects interdisciplinary fields such as biological psychology. Using the present discussion as an example, there is a mature research program addressing the mathematical properties of HR, a recent expansion in the format and accuracy of measurement devices, an increasingly complicated picture of the autonomic and cardiovascular environment, a growing number of HRV calculations available to perform, a profound spreading of interest within personal and lay scientific publications, and finally, the various application of all of the above to social science (in addition to biomedical science, exercise science, public health, and so on).

Consequent to this breadth, and the complexity established above in Factor 1, a simple experimental measurement which claims to capture a complex biological status is attractive to the applied theoretician. In other words, the idea that the analysis of HR can return a core measure of 'autonomic status' simplifies the measurement, analysis, and conception of the nervous system required to perform research on behavior. This simplification allows conceptual access to further simplifications, specifically the conception of a full-body "non-specific stress response" (Selye, 1936, 1956), the idea that spreading arousal of the SNS can be seen in all systems and subsystems. That is, if a general arousal response can be successfully induced, then it is presumably non-specifically present because of sympathetic influences on the heart, skin, viscera, vasculature and so on. The legacy of this theory mirrors the measure provided by the idea of a uniaxial HRV which reports a measurement of a single sympathetically-mediated system. It should be noted, however, that this is contradicted by a broad array of evidence (Mauss & Robinson, 2009); instead, we observe inter-coordinated responses between different sympathetically mediated sub-systems

during laboratory provocation, and specific patterns of autonomic sequelae associated with different 'arousing' stimuli (Berntson, Boysen, & Cacioppo, 1993; Kreibig, Wilhelm, Roth, & Gross, 2007).

Experimental Success

Hypotheses with any longevity are not derived in a vacuum, but from observations on top of layers of previous reasoning. The outflow of the peripheral nervous system is unambiguously central to the regulation of HR over time, and has unambiguous effects on low frequency (LF) and high frequency (HF) HRV power. Double blockade experiments (Goldberger, Challapalli, Tung, Parker, & Kadish, 2001) have long established the contributions of the SNS and PNS to HRV. Likewise, we see interrelated circulatory, endocrine, etc. changes in response to circumstances or stimuli. Thus, HRV measures a system in perpetual flux – if we have a well-constructed observation, we will almost invariably measure *some* change. This is more the case if the measurements involved are volatile (indeed, Hypothesis B shows a significantly higher relative standard error than other measurements of HRV; Heathers, 2014). This instability is the reason authors occasionally recommend against the use of any spectral analyses at all (Ng, Sundaram, Kadish, & Goldberger, 2009).

In sum, if the criteria for publishing a scientific article is simply that the measured results resolve to be statistically significant, an unstable measurement of an unstable phenomenon is an excellent vehicle for engineering differences between groups, especially considering the substantial flexibility in modern publication practices (Gelman and Loken, 2013; Simmons, Nelson and Simonsohn, 2011).

Explicit Authority / Guidelines

The most definitive statement on how HRV research should be conducted is the paper by a task force of senior researchers in electrocardiology, physiology, neurology, and similar, drawn from The European Society of Cardiology and The North American Society of Pacing and Electrophysiology. It was published multiply in *Annals of Noninvasive Electrocardiology*, *Circulation*, and *European Heart Journal*.

With regards to Hypotheses A and B, it states:

“Disagreement exists in respect to the LF component. Some studies suggest that LF, when expressed in normalized units, is a quantitative marker for sympathetic modulations, other studies view LF as reflecting both sympathetic and vagal activity. Consequently, the LF/HF ratio is considered by some investigators to mirror sympatho/vagal balance or to reflect sympathetic modulations.”

(Task Force, 1996, p.366)

This paper, a comprehensive statement of how HRV should be understood, measured, and utilized goes on to list the clinical utility of the LF/HF ratio, results of interest, and expected values the ratio might take. Definitive statements concerning how research should be conducted, typically established by senior committees or working groups within formal scientific societies, are enormously influential, thus this statement has provided an ongoing official sanction for the experimental use of Hypotheses A and B. The Task Force (1996) has more than 11,000 citations between its multiple outlets of publication, and is easily the most highly cited and enduring position statement on HRV.

While the Task Force (1996) papers remains commonly cited, it was also published prior to the objections recorded in Table 1 except for Berntson et al. (1997). This paper is the most notable for several reasons; it was published within a psychologically-focused journal almost simultaneously, had access to the same primary literature, and was the product of a similarly experienced body of experts. It also explicitly recommended against the use of the LF/HF ratio “in the absence of explicit validation over a broader range of conditions” (p.640), a prediction that seems prudent in retrospect, given the subsequent body of literature.

Implicit Authority / Precedence

Any published scientific technique may acquire a kind of self-sustaining authority in that it may continue to be acceptable simply because at some previous point it has been deemed acceptable. As Hypothesis A and B have an almost 30-year tradition of publication, it is simple to demonstrate they are ‘correct’ based on precedence. Three factors appear to maintain this reliance on implicit authority from prior publications. First, scientific works form a permanent record that is not directly updated, questioned, or invalidated. The only exceptions are where research is demonstrably incorrect or fraudulent. Second, peer review has a series of well-outlined problems (Baliatti, Goldstone, & Helbing, 2016; Henderson, 2010; Jefferson, Alderson, Wager, & Davidoff, 2002; R. Smith, 2006) and is far from an absolute statement of quality. Third, any paper rejected by peer review from an individual journal may simply be submitted elsewhere, and this may happen iteratively until it is published, a practice occasionally termed ‘journal shopping’ (Kovanis, Porcher, Ravaud, & Trinquart, 2016). A paper entirely reliant on either Hypothesis A or B has no barrier to eventual publication; Heathers (2014) found that 22 out of 97 papers (23%) using frequency analysis to report HRV used *only* calculations derived from Hypothesis B.

Dilution

Well over 100 methods exist to calculate HRV (Ramshur, 2010; Smith, Owen, & Reynolds, 2013). These methods include: (a) different categories of time- and frequency-domain measurements; (b) variations on identical methods, such as spectral analyses (Fast Fourier Transform, auto-regressive, Lomb-Scargle periodogram, pseudo-smoothed Wigner-Ville transform, etc.); (c) alterations in temporal or frequency parameters (i.e., power spectral density from 0.12 to 0.5Hz as a substitute for 0.15 to 0.5Hz, or pNN20 as a substitute for pNN50); (d) 'non-linear' measurements (a catch-all term for sample entropy, fuzzy entropy, hidden words analysis etc.), and so on. It is common for research to report multiple indices simultaneously. These indices are often highly correlated with each other, the most common example being HF (high frequency power spectral density) and RMSSD (root mean square of successive differences) which track each other closely between ($r=0.91$; Massin, Derkenne, & von Bernuth, 1999) and within ($r=0.883 - 0.977$; Otzenberger et al., 1998) subjects.

Dilution reduces the importance of any reported individual measure – if a dozen inter-related HRV measurements are reported, it is less problematic to include one which might have poor evidentiary capacity, as its effect on the overall conclusion of the paper is diminished. A recent example is Dantas et al. (2017) who cite both the problems listed in Table 1, but also provide reference values congruent to both hypotheses “as new insights may emerge in future work.”

Personal Investment

It is entirely human nature to defend deeply held convictions. However, when those convictions are about scientific assertions and not supported by the best available evidence, it is

an impediment to progress. One conception of this tendency in scientists, as inflexible defenders of favored ideas, is often referred to as 'Planck's Principle' subsequent to Max Planck's famously bleak view of scientific progress ("new scientific truth does not triumph by convincing its opponents ... but rather because its opponents eventually die"; p.33-34, Planck, 1949). Planck's statement was not intended to be axiomatic, and was certainly not true of either Planck himself or his close contemporaries (e.g. Helmholtz, Heisenberg) who displayed fundamental shifts in thinking during their working lives (Blackmore, 1978). However, it is not without evidence – the death of scientific 'superstars' within the life sciences lead to a noticeable change in the pattern of publication in favor of their non-collaborators (Azoulay, Zivin, & Wang, 2010). Within the context of our hypotheses in question, as recently as 2012, some of the original authors have offered a limited defense of the hypotheses in question, consisting primarily of a restatement of the results of the original experiments, and a renewed call for further research (Pagani, Lucini, & Porta, 2012). This defense is maintained without addressing the substantive objections referenced in Table 1.

Conclusion

This paper outlines an example how a specific scientific hypothesis can attain parallel status – abandoned in basic research, yet alive in applied research. The hypotheses in question are of direct relevance to fields including biological psychology, psychophysiology, and social neuroscience that use physiological measurements to answer applied questions with broader social scientific relevance. While this is presented as a case study, it is certainly not an isolated example of psychological research failing to correctly situate itself in physiological evidence – for instance, one heavily cited theory of self-control posited that a dip in blood glucose due to mental effort meant peripheral glucose and its manipulation formed a reasonable physiological proxy of self-

control (Gailliot and Baumeister, 2007). This is at odds with basic neurophysiology; the brain's energy consumption, measured by direct methods, does not in any way utilize sufficient glucose for the model to be correct, nor does it change during strenuous tasks (e.g. Madsen et al. 1995). Likewise, investigations into 'power posing' (adopting an expansive, powerful posture) were originally posited to increase testosterone levels (Carney, Cuddy & Yap, 2010). However, this result overlooks some basic endocrinological facts, specifically that (a) testosterone levels of men and women were aggregated and analyzed with gender as a covariate, but as they are produced by almost entirely different biological processes, their absolute changes as analyzed are non-equivalent (i.e., a change in male testosterone levels is not physiologically equivalent to a similar change in female testosterone), and (b) salivary testosterone is an extremely variable predictor of free testosterone in women, the bioactive component presumably capable of changing dominance behavior ($r=0.37$; Granger et al., 2004). Other applied sciences have long dealt with how similar measurement problems should be approached (e.g., Fesselstein, 1985).

It should be noted immediately that the hypotheses under discussion here were never *scientifically* problematic. When proposed subsequent to demonstrations that sympathetic outflow and HRV covaried closely with orthostatic tilt angle (e.g., Pagani et al., 1984, 1986), these hypotheses were reasonable, grounded in well-collected physiological observations, and capable of being falsified, i.e., able to generate specific and testable predictions. Since then, a broad base of further evidence was amassed within human cardiac, circulatory, and autonomic physiology such that the hypotheses do not work as described. These were important and popular metrics, they attracted appropriate scrutiny, and were subsequently discarded. The above reflects well on the scientific process within basic research. The present ensuing period of 'life after death' within applied research does not.

The best specific advice this paper can offer is to encourage research into the assumptions and efficacy of other techniques which capture any version of cardiac sympathetic activity. If we accept the assumption that researchers who wish to move past HRV-based techniques to approximate cardiac SNS activity are already familiar with ECG recording, four related and similarly non-invasive techniques exist within cardiovascular signal analysis which may be of value. First, the use of an impedance cardiograph to approximate pre-ejection period (Newlin & Levenson, 1979), a systolic time interval of contractility largely controlled by the SNS. It requires the use of an ECG plus an additional device to measure thoracic impedance (Van Dijk et al., 2013). Second, the reproduction of the above (i.e., measuring relevant systolic time intervals) by analysis of heart sounds (Carvalho et al., 2009; Paiva et al., 2012). Third, the analysis of T-wave height (Heslegrave & Furedy, 1979; Muller, Schandry, Montoya, & Gsellhofer, 1992; Rau, 1991), as reflective of ventricular repolarization. Fourth, the analysis of skin sympathetic nervous activity (SKNA; Doytchinova et al., 2017; Jiang et al., 2015), where the high-frequency 'noise' of a very fast sampled ECG (i.e., 150-1000Hz) is reflective of sympathetic innervation of the skin. These techniques are heterogenous and have various degrees of support, but all rely on less ambiguous observations and better-supported causal reasoning than retrieving sympathetic activity from HRV. They are also similarly non-invasive, and thus can be deployed in humans (i.e., can be obtained within a typical psychological laboratory) with the same ease as any other biometric measurement.

Past the above recommendations, how to address the broader publication environment is less clear. If providing the information as stated in Table 1 was sufficient to change research practice, then the present discussion would only be of historical interest rather than immediate concern. Also unclear is what urgency should exist around this issue. For instance, should a meta-scientific framework exist which could alter publication behavior? To retract the original papers

is inappropriate as there is no suggestion of wrongdoing. Indeed, retraction itself may not even be effective – the recent retraction of papers by a researcher convicted of data fabrication did not appear to dramatically change citation habits of the affected papers (Bornemann-Cimenti, Szilagyi, & Sandner-Kiesling, 2016; Teixeira da Silva & Bornemann-Cimenti, 2017). In light of the above, it seems entirely more appropriate that the onus to recognize and ameliorate the problem outlined herein lies with present authors and journals within psychology, and not with the original theory or authors.

There are some other possibilities for field-wide clarification. PubMed Commons and PubPeer both offer platforms for the scientific public to visibly annotate published articles. It may also be possible to automate responses on such platforms (Nuijten, Hartgerink, Assen, Epskamp, & Wicherts, 2015) – a strong statement could be made by automatically annotating all published articles that base conclusions on methods which overwhelmingly lack support. Psychophysiological journals (or the scientific societies which maintain them) might consider adopting analytical or editorial standards for analyses which are applied pre-publication and compel researchers to consider alternative methods. This sets a precedent, however, if an idea exists along a balance of evidence, at what point should a community of scientists compel others to use (or avoid) a certain theory? What represents a sufficient standard of proof in such a situation? Such a standard would invariably be high. Does the evidence presented here, as extensive as it is, meet such a standard?

Also unclear is the importance of the problem. Crises in and around research bring with them their own specific urgencies. The failure of a drug already brought to market due to unacceptable death rates (Cerivastatin, Vioxx, Mibefradil, etc.) demands an immediate and coordinated research, regulatory, and legal response. However, the failure of a well-accepted social

psychological effect does not (see recently, for instance, the treatment of various priming effects; Shanks et al., 2015) – the response to such an issue is certainly less urgent than one causing widespread mortality. The danger of the present status of Hypotheses A and B to biological psychology is presumably somewhere between these examples. While a formal literature-wide updating of standards of publication would be a substantial task, including a variety of associated fields and sub-fields with separate conventions for publication standards, maintaining a central base of scientific knowledge as bedrock for further investigation is crucially important regardless of the area of study. The alternative, if not actively dangerous, increases the probability that researchers' time and public money will be poorly deployed.

Having previously declared an obituary for the hypotheses outlined here (Heathers, 2012), it is a curious task five years later to consider their 'life after death.' While basic research necessarily progresses slowly, applied research will move slower still if the factors outlined in our case study are not appropriately acknowledged and satisfactorily addressed.

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