### Accepted Manuscript

Combining field work and laboratory work in the study of financial risk-taking

John Coates, Mark Gurnell





Please cite this article as: John Coates, Mark Gurnell, Combining field work and laboratory work in the study of financial risk-taking. The address for the corresponding author was captured as affiliation for all authors. Please check if appropriate. Yhbeh(2016), doi: 10.1016/j.yhbeh.2017.01.008

This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

### Opinion

### Combining field work and laboratory work in the study of financial risk-taking

John Coates<sup>1</sup>, Mark Gurnell<sup>2</sup>

<sup>1</sup>Dewline Research, London, W8, United Kingdom; <sup>2</sup>Wellcome Trust-MRC Institute of Metabolic Science & NIHR Cambridge Biomedical Research Centre, University of Cambridge & Addenbrooke's Hospital, Cambridge, CB2 0QQ, United Kingdom

#### Abstract

A contribution to a special issue on Hormones and Human Competition. Financial markets are periodically destabilized by bubbles and crashes during which investors display respectively what has been called 'irrational exuberance' and 'irrational pessimism'. How can we best study these pathologies in competitive and risk-taking behaviors? In this article, we argue that a science of risk-taking and of the financial markets needs to draw heavily on physiology and especially endocrinology, due to their central roles in moderating human behaviour. Importantly, this science of competition and risk requires the same spectrum of research protocols as is found in mature biological and medical sciences, a spectrum running from field work conducted within financial institutions themselves to more controlled laboratory studies, which permit cause to be distinguished from effect. Such a spectrum of studies is especially important for translational behavioural science.

### Highlights

- Cycles in financial markets tend to overshoot levels that could be justified by current earnings

- Risk preferences appear to shift pro-cyclically

- Endocrine systems play a role in shifting risk preferences

- Understanding shifting risk preferences and market instability requires a combination of field and laboratory work.

### Key words

Testosterone. Cortisol. Financial market. Bubble. Financial crisis. Risk preference. Exuberance. Pessimism.

#### Introduction

The financial markets present us with the largest and most intense competitive forum ever constructed. Here the outcome of competition is, according to classical economic theory, an optimal allocation of capital to the projects with the highest returns and, thus, an increase in global prosperity. Financial markets can admittedly be volatile and do cycle between 'bull' and 'bear' markets. These cycles have an average period of five to six years and can be considered healthy responses to the ebb and flow of credit, and to technological and economic opportunity. Unfortunately, the cycles tend to overshoot (Shiller & Page, 1981), occasionally to such an extreme that they threaten the stability of the global economy.

For example, bull markets can morph into bubbles, in which investors display what has been termed 'irrational exuberance' - an unrealistic assessment of expected returns and of their own ability to predict the future; in contrast, bear markets can morph into financial crises, in which investors display 'irrational pessimism' - an almost complete aversion to risk. During bubbles and crashes investors typically react to price changes in a manner which is precisely the opposite to what economics would predict: the higher securities' prices rise, the more investors buy them; the lower prices fall, the more investors shun them. Indeed, during the Credit Crisis of 2008-9 some argued that the markets had been pulled into a singularity in which the laws of economics no longer held true (Sornette and Cauwels, 2015). Irrational exuberance and pessimism in our competitive and risk-taking behaviours thus contribute significantly to instability in our financial system.

So how can we gain a better appreciation of the factors that underlie these almost pathological forms of risk-taking? In this article, we propose an answer to this important question, reasoning that a science of risk-taking in the financial markets should not ignore the basic principles of physiology, and especially endocrinology, given their central roles in moderating human behaviour. Importantly, this science of risk should employ the same robust research tools and protocols as are commonly used in more mature biological and medical sciences – a spectrum which combines field work (in this case within financial institutions) with more controlled laboratory studies (that allow distinction between 'cause' and 'effect').

#### Behaviour during extreme market volatility

Research on market phenomena occurring during non-volatile times can be pursued successfully using commonly employed economic tools, such as axiomatic modelling, computerized decision

making tasks, questionnaires, etc., and with participants recruited from, for example, a student body or the wider population. But research on market phenomena that occur in what are commonly referred to as the 'the tails' (Fig. 1), in other words, when markets move three or more standard deviations – as is the case with bubbles and crashes – faces several challenges.

To begin with, standard research tools may not be particularly well-suited to studying these extremes. For example, during tail events, such as the singularity of the 2007-09 Credit Crisis, the pressures felt by investors and traders were so intense that it is almost impossible for researchers to recreate these in a laboratory setting. Appreciating what a trader really experiences in the 'heat of battle' is challenging enough, but then recreating this in a group of volunteers who know that the task in hand is simulated (and they can therefore withdraw to safety at any stage without untoward consequence), means that the true range of physiological changes and emotions that the traders experience in real life are likely to be poorly reproduced in the artificial setting.



**Fig. 1.** Normal distribution of stock market returns. X-axis units denote standard deviations from mean; Y-axis denotes probability density.

During bubbles, for example, investors and traders (many of whom are fresh out of college with no prior experience of making a lot of money) start enjoying above-average profits, their incomes spike to levels they have never before enjoyed, and they glimpse the possibility of real wealth. It is of course almost impossible to simulate this degree of euphoria with the payouts commonly

made to participants in behavioural experiments (which are typically small amounts, and rarely exceed a few hundred dollars.)

During financial crises and market crashes the same investors and traders suffer losses greater than anything they have ever contemplated; they face the loss of their jobs, even personal bankruptcy, and suffer the social shame of having to curtail the lavish lifestyle that accompanied their increased wealth during the prior bubble. These financial crises are particularly powerful events, with the daily news reports of failing financial institutions and hasty government and central bank interventions amplifying the fear spreading throughout the financial community. Unless one has experienced these tail events, or has observed first hand investors and traders caught up in them, it can be difficult to fully understand how profoundly they affect a person's willingness and ability to take risks. Indeed, such challenges are inherent in any laboratory study of simulated competition (e.g. when studying the performance of elite athletes, it is difficult, if not impossible, to reproduce the uniqueness of real competition and the heat of the moment).

A second difficulty in researching tail events stems from the existing tools that are commonly used to understand the markets. Most models in economics and finance are built upon the foundational assumption that risk preferences are a stable trait, much like a person's height or eye colour (Sigler and Becker, 1977; Luigino and Sugden 2007). So pervasive is this assumption that it is even found in the indices of risk appetite constructed by central banks (Gai and Vause, 2006; European Central Bank 2007). It did prove useful during the formulation of Neo-Classical economics and the core models of formal finance, such as the Capital Asset Pricing Model, because these models were built on the axioms of rational choice. Stable preferences were required because they ensured that choices were consistent.

The models built upon this foundational assumption were elegant and highly influential. They even retained a core of commonsense in that they were informed by the not unreasonable belief that when people manage their money and try to maximize their wealth they act rationally. Unfortunately, however, this assumption does not seem compatible with the observed behaviour of investors during tail events. During bubbles investors appear to become more risk seeking and during crashes more risk averse. Indeed, since the credit crisis several studies have emerged (based on brokerage data) suggesting that risk aversion did increase during and after the crisis (Guiso 2013; Smith and Whitelaw, 2009; Cohen et al, 2015).

A third, and related, difficulty impairing our understanding of the markets is that most models in economics and finance assume risk-taking in this context to be a purely cognitive activity, i.e., that it can be understood by studying just the decisions themselves (employing tools such as logic, information theory, game theory, cognitive psychology, etc), but without reference to the physical changes taking place in the body of the risk taker. This assumption represents a profound misunderstanding of human biology and of the physiological pathways connecting body to brain. Body and brain evolved together and operate together. This unity of body and brain is evident when we take risk in, say, sports or war, because at these times the changes in our bodies – the quickened heart rate, increased breathing and blood pressure, the flood of both anabolic and stress hormones into the blood stream - is so overwhelming that we are readily aware of them (Coates, 2012).

It has been the hypothesis of the current authors that these somatic changes are also – and indeed especially – important to risk-taking in the financial markets and for understanding the cycles of bubble and crash. We have found that risk preferences do in fact shift, that the shifts are large, and that the shifts are caused by alterations in physiology (Kandasamy, 2014). To study changing risk preferences, we have sought to apply insights and research protocols drawn from the disciplines of endocrinology and sports physiology to the world of finance. Importantly, our findings confirm that physiological changes, in particular fluctuations in classical endocrine pathways (anabolic and catabolic), are major determinants of changing risk preferences.

#### Hormones and risk-taking

Findings from sports physiology and from animal models such as John Wingfield's Challenge Hypothesis (Wingfield et al, 1990) suggest that anabolic hormones can prepare the body and brain for competition. For example, testosterone promotes muscle hypertrophy and increases the level of haemoglobin in the blood and thus its capacity to carry oxygen (Bhasin et al., 2010). In the brain testosterone has been linked to increased confidence and search persistence in small mammals (Andrew and Rogers, 1972; Archer, 1977), and preference for novelty in both animal and human studies (Maattanen 2013; Boissy and Bouissou, 1994; Hermans et al, 2006); it interacts with dopaminergic circuits thereby making competitions euphorogenic (Schroeder and Packard, 2000; Frye et al, 2002); and it has been reported to increase appetite for risk, although some researchers contest such cause and effect (Booth et al, 1999; Apicella et al, 2014; Apicella et al, 2015; Dreber, 2009).

Researchers in biology have postulated that the above mechanisms mediate the remarkable and related phenomenon known as the 'winner effect' in which an animal winning a competition enjoys an increased chance of winning its next competitive or agonistic encounter (Dugatkin, 1013; Rutte et al, 2006; Lehner et al, 2011; Chase et al, 1994; Hsu and Wolf, 2001; Geniole et al, 2017). Researchers have catalogued this phenomenon among a wide range of taxa, and a subset of these researchers, when looking for the underlying mechanism, have found that testosterone rises in the winner of a competition while falling in the loser. Furthermore, the winner's androgenic priming gives it an edge in the next round of competition, leading to a feedback loop in which the very act of winning raises testosterone, which in turn contributes to further success (Oyegbile and Marler, 2005; Earley et al, 2013; Trainor et al, 2004; Oliveira et al, 2009; Fuxjager et al, 2010). Evidence of a testosterone-mediated winner effect has also been described in human male competitors (in both field and laboratory settings), although such an effect has not been universally observed (Booth et al, 1989; Carre and Putnam, 2010; Mazur et al, 1992; Carre et al, 2013; Zilioli and Watson, 2014).

It is also likely that such a mechanism of empowerment could not continue indefinitely. In this context, it is interesting to speculate that testosterone, like several other hormones, may display an inverted U-shaped dose response curve, meaning that beyond an optimal (testosterone) level for a given competition, any further increase may actually impair performance. Evidence to support such a hypothesis is provided by animal studies in which elevated testosterone (i.e. raised beyond levels required for mating or normal territoriality) can encourage animals to fight too often, stray into the open, patrol areas too large, neglect parenting duties, and deplete fat/energy stores, all of which lead to increased vulnerability and even mortality (Wingfield et al, 2001; Dufty, 1989; Marler and Moore, 1988; Beletsky et al, 1995). At these elevated levels of testosterone, effective risk-taking morphs into risky behavior. Further studies, examining risk-taking behaviours across a spectrum of (exogenously manipulated) testosterone concentrations in animal models would help to confirm the validity of the proposed U-shaped dose response curve.

Several years ago, we developed the hypothesis that a variant of the winner effect is at work in the financial markets, during both individual winning streaks and more broadly during bubbles, which are market-wide winning streaks. At these times, traders and investors make above-average profits; their victories raise testosterone levels which, in turn, increase risk appetite and trade size (Coates and Herbert, 2008; Coates and Page, 2009; van Honk et al, 2004; Reavis and Overman, 2001; Pope et al, 2000; Stanton et al, 2011). Eventually, however, traders go beyond the peak of

their inverted U-shaped response curve and any further increase in testosterone begins to impair risk-taking. Traders become over-confident and place bets in ever increasing size, with everworsening risk-reward trade-offs, until eventually their trades go wrong and they suffer such large losses that they lose more money than they made on the winning streak.

We also hypothesized that during bear markets – which so often spiral into financial crises and crashes - catabolic and stress mechanisms come to dominate risk-taking behaviour. During all competitive and risk-taking situations stress hormones such as adrenaline and cortisol promote an anticipatory arousal (Dickerson and Kemeny, 2004; Dallman and Bhatnagar, 2010). They prepare a person metabolically for impending physical activity, breaking down energy stores [liberating glucose in liver and muscle, free fatty acids in adipose tissue (fat depots)] to provide the fuel needed for this work, (i.e. driving catabolic processes throughout the body.) Acute increases in stress hormones raise blood glucose levels, increase heart rate and blood pressure, and inhibit bodily functions not required for immediate survival, such as digestion and reproduction. In the brain, cortisol (which crosses the blood-brain barrier) heightens recall of emotionally relevant memories (Lupien et al, 2002) and, by interacting with dopaminergic circuits, contributes to making acute risk-taking euphorogenic (Piazza and LeMoal, 1997; Sarnyai et al, 1998; Piazza et al, 1993; Putman et al, 2010).

However, the effects of an acute (i.e. short-lived) rise in cortisol can differ dramatically from those of a chronic (i.e. sustained) elevation. When increased cortisol levels persist for days or weeks they can contribute to the development of gastric irritation (even frank ulceration), abdominal (visceral) obesity, insulin resistance and type 2 diabetes, abnormal blood lipid profiles, cardiovascular disease (Anagnostis et al, 2009) and impaired immune function (McEwen et al, 1997). In the brain, chronically elevated cortisol impairs attentional control (Liston et al, 2009) and behavioural flexibility (Dias-Ferreira et al, 2009; Schwabe and Wolf, 2009); it promotes anxiety (Korte, 2001; Corodimas et al, 1994), a selective recall of disturbing memories (Erikson et al, 20003), a tendency to find danger where none exists (McEwen, 1998), even depression and learned helplessness (Sapolsky, 2000; Kademian et al, 2005). Given this suite of effects, it seemed reasonable to us to hypothesise that chronically elevated cortisol levels would also promote increased financial risk aversion.

We thus developed an endocrine-based model to explain why financial market cycles tend to overshoot, become unstable, and require government and central bank intervention to restore

stability. During bull markets a financial variant of the winner effect causes risk preferences to shift towards greater risk seeking: the rising market leads to above-average profits; testosterone levels rise; confidence and trade size increase thereby contributing, on average, to increased profits. However, at some point in this upward spiral testosterone levels exceed the peak of the dose-response curve and begin to promote the irrational exuberance that pushes a bull market into a bubble. Once the bubble bursts and a bear market ensues, the increased uncertainty and volatility raises cortisol levels, and as this stress response persists and becomes chronic the cortisol promotes risk aversion and the irrational pessimism that pushes a bear market into a crash. In short, our endocrine system contributes to pro-cyclical shifts in risk appetite.

#### Field work and laboratory studies – a complementary approach

How do we test this model? For that matter, how do we test any model of shifting risk preferences in the financial markets? The classical scientific approach demands a controlled laboratory study; but ensuring that such a study is relevant and meaningful first requires observations in the field to help inform study design (SciBytes, 2013). On a trading floor, for example, this field work could involve monitoring risk takers' physiology (their hormones, cardiovascular system, etc) as well as individual performance data (their profits and losses (P&L), market volatility, etc.). In fact, the model as presented here was not worked out *a priori* but was itself the result of extensive field work we conducted on trading floors in both New York and London before the 2008 financial crash, observing traders as they took real positions in the markets.

Field work is an essential first step in scientific discovery. It permits researchers to observe phenomena that are unexpected or anomalous for existing theory, much like observing, for example, that the orbit of Mercury was other than that predicted by Newtonian mechanics, or that the geographical and fossil records could only be explained by a new theory of plate tectonics. In behavioural sciences, field work also presents researchers with an opportunity to identify problems in the private sector that would benefit from scientific study. In other words, field work provides external ecological validity for research. Field work is particularly important when researching financial market instability because, as highlighted above, it is difficult to replicate in a laboratory setting the pressures of real high-stakes risk-taking, or to predict by means of theory alone the behaviour resulting from this pressure.

Field work, however, has its drawbacks. To begin with, often it cannot establish causation once a correlation has been observed; and it can be difficult to control for potential third confounding variables. This does not mean that field work produces nothing more than 'mere correlation'. Observation and correlation are the first steps in scientific discovery so should not be dismissed. Moreover, if one variable in a correlation is separated temporally from another, then this can provide a strong pointer to the primary aetiological factor (although third party confounding factors must still be considered). For example, when testing the winner effect hypothesis we noted in one study that when traders experienced high testosterone in the morning they made more money in the afternoon (Coates 2008). Here the high testosterone occurred before the trading profits, so we could argue with reasonable confidence that the testosterone predicted the profits.

Importantly, the external validity provided by field work enables it to guide researchers as to where they should be focusing their attention in the laboratory setting. Laboratory work then permits the researchers to achieve internal validity, in other words, to control the variability in one parameter and to observe its effects, thereby confirming or excluding causation. In the laboratory additional variables can also be more readily controlled for; and the experimental protocol can be repeated more easily.

Combining field and laboratory work in this way is common in biology and medicine. Instructive examples are found in the work of Robert Sapolsky and colleagues on the neurophysiology of the stress response (Sapolsky, 2004), and among many of the contributors to this special volume such as John Wingfield, Catherine Marler, and Coren Apicella. Their combination of field and laboratory work is, however, not so common in economics and finance (Eisenegger and Naef, 2011). It is unfortunate that this should be so, for reasons already outlined. But there is a further reason in the case of financial risk-taking: the trade-off between the relevance of field work and the rigor of the laboratory is not as dramatic in financial phenomena. The authors' experience has been that a trading floor presents a rare research venue where you can find the behaviours and pressures of the real world combined with elements of reproducibility/controllability that are more typical of a laboratory setting.

There are various types of trading floors. The most well-known are the floors of stock exchanges, where brokers jostle and yell out orders. These have mostly been replaced today by computerized order matching systems, but a few remain, most notably in New York and Chicago. More

common are the trading floors of individual banks and trading firms. These may appear more sedate than the floors of exchanges, but the competition is nonetheless intense and the risks being taken are huge. On these floors, traders and asset managers sit at the same desk every day, surrounded by banks of computer screens on which are displayed real time news feeds and security prices. The traders have their allotted markets to trade and fixed risk limits; and in some sense they do the same thing every day – i.e. it is repeatable. And in that seat they experience in a compressed period of time levels of reward that are considerably higher than most people will experience in their entire lives and, equally intense, highly elevated levels of uncertainty and stress. The trading floor thus offers a rare venue in which to combine the rigor of the laboratory with the relevance of the field.

#### Volatility and the stress response

An example of research into financial risk-taking conducted along the methodological lines outlined here may be instructive. In one of our studies on a trading floor we set about testing for a testosterone-mediated winner effect in traders. We also screened for something akin to a cortisol-mediated loser effect in which, we hypothesized, that trading losses might be amplified by rising levels of the stress hormone cortisol. But this latter hypothesis was not confirmed by the data: losing money did not seem to have a large effect on the traders' cortisol levels (Coates and Herbert, 2008). This was surprising. It is possible that the risk management at this firm was good enough to stop traders from losing much money, and that larger losses than we observed would have triggered a rise in cortisol. But what we observed instead was that the traders' cortisol levels were remarkably sensitive to the variance in their profits and losses (P&L), and to volatility in the market. This opened up a line of enquiry that we had not previously considered.

On reflection, this finding is consistent with what is already known about situations that are associated with changes in cortisol status. For example, cortisol levels increase as a result of insult/injury, but a similar rise can be seen in situations where no harm has actually occurred, but only anticipated (Hennessy and Levine, 1979). Here the cortisol surge is part of a preparatory stress response, much like an early warning system that places our physiology on high alert. These situations are ones of novelty, uncontrollability, and uncertainty (Hennessy and Levine, 1979). Each of these is a permanent feature of the financial markets, so in retrospect it should not have come as a surprise that traders' cortisol levels were as volatile as we found.

We were able to proxy uncontrollability in the experiences of traders by using the variance of their P&L. A trader in control will have a relatively constant P&L, but as he or she loses control, making money one day, losing it the next, the variance of their P&L rises. We found that cortisol levels of the traders rose with the variance of their P&L ( $R^2$ =0.47, p=0.002) (Coates & Herbert, 2008). We were also able to proxy the uncertainty faced by the traders by using the volatility of the markets: the higher the volatility, the wider the dispersion of future prices traders must use in predicting returns. For example, the VIX is an index of volatilities on U.S. stocks and it has been called the Fear Index as it is a sensitive indicator of uncertainty among the financial community. During the Credit Crisis of 2008-9 the VIX spiked from 12% to over 70%. In our study we found that over a two week period mean daily cortisol levels on the trading floor rose and fell in line with volatility in the German Bond market (which was the market in which all traders were placing their bets); and that the hormones and volatility were highly correlated ( $R^2$ =0.86, p=0.001). This remarkably high correlation has since been replicated in as-yet unpublished studies set in different markets and firms (Coates & Gurnell, unpublished data).

Another notable finding in this study was that cortisol levels in the traders rose 68% over an eight-day period (Coates 2008) – i.e. the traders experienced sustained (chronic) elevation of cortisol (which contrasts with the transient rise that might be seen in an acute (short-lived) stress response (Schultz et al, 1998). The question thus arose: is this observation important? Did the increase in cortisol itself affect the traders' risk-taking behaviour?

To answer these questions, we took our findings in the field back to the laboratory. Using a double-blind, placebo controlled, cross-over study design (i.e. a format commonly used to assess the efficacy and safety of a new medicine/technology before it can be considered for use in routine clinical practice), we examined the effects of both acute and more sustained elevations in cortisol (Kandasamy et al, 2014). One group of participants received synthetic cortisol, in the form of hydrocortisone tablets, for eight days, followed by a washout period, then eight days of placebo tablets. A second group of participants followed the reverse schedule, i.e. placebo-washout-treatment. A third sub-group received placebo-washout-placebo to test for learning effects on the behavioural tasks (i.e. changes occurring independent of cortisol status). The dosing regimen was designed to replicate the natural increase in cortisol levels that we had previously observed in the traders. Previous studies that have examined the effects of changes in endocrine status on psychological and physical parameters have more often than not fallen into the trap of

using supraphysiological dosages that immediately call in to question the relevance of their findings to normal physiology.

The study protocol we designed therefore had several key features:

- Treatment was tailored to individual participants, i.e. we avoided a 'one size fits all' approach (whereby everyone receives the same dose), and instead based dosing schedules on body weight (thereby adopting a well-validated algorithm which has been used in clinical practice to calculate daily cortisol requirements in patients with adrenal failure (Mah et al, 2004), and adjusted to raise subjects cortisol levels by 68% over the eight days of the study
- 2) Dosing schedules induced a sustained rise in cortisol throughout the study period, abolishing the normal diurnal (circadian) rhythm of cortisol production, and reproducing the profile observed in stressed individuals; to achieve this, we used a multi-dosing regimen (thrice daily – in the morning, early afternoon and evening)
- Participants collected salivary samples at home at several time points during the 8-day study period – thereby allowing us to assay for cortisol and confirm compliance with the dosing protocol
- 4) The behavioural tasks were incentivized so that the participants tried at all times to make as much money as possible on the trading tasks. To achieve this, subjects were offered the possibility of making over \$425 on the study.

At the beginning and end of each phase (active and placebo, placebo and active, or placebo and placebo), participants played a computerized gambling task in which they had to choose between lotteries offering different combinations of monetary returns and probabilities. We succeeded in raising participants' cortisol levels by an average of 69% over the eight days of the study, thereby replicating almost exactly the levels observed in traders. Crucially, this had the effect of shifting participants' risk preferences so that they preferred safer lotteries, ones with lower expected returns but with lower variance of returns. In other words, they became significantly more risk averse, with an effect size that was large (risk aversion increased by 44%). In contrast, a short-lived elevation in cortisol (assessed by repeating the computerized task on the first day of the study 90 minutes after taking the first hydrocortisone tablet) had no discernible effect on risk preferences (Fig. 2.) (Kandasamy et al, 2014).



**Fig. 2**. **A**. sample lottery used in computer task.. **B**. Changes in the expected return and variance of the chosen lotteries. Under chronically raised cortisol participants chose safer lotteries (i.e., ones with lower payoffs and a lower variance of possible payoffs). Results are shown as means  $\pm$  SEMs. (Reproduced from Kanadasamy et al, PNAS, 2014.

In this series of studies, we began with the unexpected finding that trader cortisol levels tracked volatility closely over a period of several days. Subsequent laboratory work showed that these hormone changes can affect a core parameter of risk-taking, the traders' risk preferences. Contrary to the assumptions of much formal economics and finance, it appears therefore that risk preferences do shift; the shifts are large; and it is our physiology, in this case our HPA axis, that is bringing about these shifts.

Specifically, the findings from our field work provide clear evidence that extended periods of high uncertainty and volatility raise cortisol levels; and the findings from our lab studies show that, in time, this increased cortisol raises risk aversion itself. When does volatility in the financial

markets rise the most? During bear markets. So just when the markets are becoming cheap and should be bought, and when the markets are most in need of support, the financial community becomes more risk averse. This risk aversion accelerates the downward movement of the markets. Cortisol mediated risk-aversion may therefore be destabilizing our financial markets.

#### Conclusion

In this article, we have argued the case that behavioural and policy sciences such as finance and economics should combine field and laboratory work in a manner similar to that employed in other scientific disciplines (e.g. biology, medicine). If research questions are based on observations made in the real world (i.e. in financial institutions and in the markets), and then subjected to rigorous scientific analysis in the laboratory, we can be confident that some of the hitherto unexplained fluctuations in the markets, with wide-reaching ramifications for the global economy, will begin to be better understood.

In the case described here, we believe the demonstration of hormone-induced changes in risk preferences could help financial institutions, central banks and regulators recognize that an underappreciated and destabilizing factor in the financial markets is the instability and pro-cyclical nature of risk preferences.

Knowing that risk preferences shift under the influence of physiological systems could then help inform novel policy developments (Coates, 2012). For example, we know that stability in the markets requires a diversity of opinions – when some people sell a security you need others to buy it, otherwise market moves become exaggerated. Given the influence of physiology on risk preferences, it is tempting to speculate that stability may be served by greater physiological and endocrine diversity in the markets, a goal that might be achieved for example by employing a more even mix of men and women, young and old in financial institutions.

### Acknowledgements

M.G. is supported by the National Institute for Health Research Cambridge Biomedical Research Centre.

octering with 

### References

Anagnostis P, Athyros VG, Tziomalos K, Karagiannis A, Mikhailidis DP, 2009. Clinical review: The pathogenetic role of cortisol in the metabolic syndrome: A hypothesis. J Clin Endocrinol Metab 94:2692–270.

Andrew RJ, Rogers LJ, 1972. Testosterone, Search Behaviour and Persistence. Nature 237:343-346.

Apicella CL, Dreber A, Mollerstrom J, 2014. Salivary testosterone change following monetary wins and losses predicts future financial risk-taking. Psychoneuroendocrinol 39:58-64.

Apicella, C, Carré J, Dreber A, 2015. Testosterone and economic risk-taking: A review. Adaptive Human Behavior and Physiology, 1: 358–385.

Archer J, 1977. Testosterone and persistence in mice. Anim Behav 25,479-488.

Beletsky, L., Gori, D., Freeman, S, Wingfield, J, 1995. Testosterone and polygyny in birds. Curr. Ornith. 12, 141.

Bhasin, S., Cunningham, G. R., Hayes, F. J., Matsumoto, A. M., Snyder, P. J., Swerdloff, R. S., & Montori, V. M. (2010). Testosterone therapy in men with androgen deficiency syndromes: an Endocrine Society clinical practice guideline. *The Journal of Clinical Endocrinology & Metabolism*, *95*(6), 2536-2559.

Boissy A, Bouissou MF, 1994. Effects of androgen treatment on behavioral and physiological responses of heifers to fear-eliciting situations. Horm Behav 28, 66-83.

Booth A, Shelley G, Mazur A, Tharp G, Kittok R, 1989. Testosterone, and winning and losing in human competition. Hormones and behavior 23, 556-571.

Booth, A., Johnson, D. Granger, D, 1999. Testosterone and men's health. J. Behav. Med. 22, 1–19.

Carre JM, Putnam SK, 2010. Watching a previous victory produces an increase in testosterone among elite hockey players. Psychoneuroendocrinol 35, 475-479.

Carré, J. M., Campbell, J. A., Lozoya, E., Goetz, S. M., & Welker, K. M. (2013). Changes in testosterone mediate the effect of winning on subsequent aggressive behaviour. Psychoneuroendocrinology, 38(10), 2034-2041.

Chase, I.D., C. Bartolomeo, Dugatkin, L. 1994. Aggressive interactions and inter-contest interval: how long do winners keep winning? Animal Behav, 48, 393-400.

Coates, J.M. Herbert, J, 2008. Endogenous steroids and financial risk-taking on a London trading floor. P NATL ACAD SCI USA, 105, 6167-6172.

Coates, J.M. Page, L, 2009. A note on trader Sharpe Ratios. PloS One, 4, e8036.

Coates, J.M., 2012. The Hour Between Dog and Wolf: How Risk-Taking Transforms Us, Body and Mind. Penguin - Random House, New York.

Cohn, A., et al. 2015. Evidence for Countercyclical Risk Aversion: An Experiment with Financial Professionals. Am Eco Rev. 105, 860.

Corodimas KP, LeDoux JE, Gold PW, Schulkin J, 1994. Corticosterone potentiation of conditioned fear in rats. Ann N Y Acad Sci 746,392–393.

Dallman M, Bhatnagar S, 2010. Chronic stress and energy balance: Role of the hypothalamopituitary-adrenal axis. Comprehensive Physiology (Wiley, New York), pp 179–210.

Dias-Ferreira E, et al., 2009. Chronic stress causes frontostriatal reorganization and affects decision-making. Science 325, 621–625.

Dickerson, S, Kemeny, M. 2004. Acute Stressors and Cortisol Responses: A Theoretical Integration and Synthesis of Laboratory Research. Psycholog Bull. 130, 355-391.

Dreber, A. 2009. Determinants of economic preferences. Stockholm School of Economics.

Dufty, A.M., 1989. Testosterone and survival: a cost of aggressiveness? Horm Behav. 23, 185-193.

Dugatkin LA, 2013. Principles of animal behavior (W.W.Norton, New York ).

Earley RL, Lu C-K, Lee IH, Wong SC, Hsu Y, 2013. Winner and loser effects are modulated by hormonal states. Frontiers Zool 10,6-6.

Eisenegger, C., Naef, M. 2011. Combining Behavioral Endocrinology and Experimental Economics: Testosterone and Social Decision Making. *J Visualized Experiments* 49, 2065.

Erikson, K., Drevets, W. Schulkin, J. 2003. Glucocorticoid regulation of diverse cognitive functions in normal and pathological emotional states. Neurosci. Biobehav. Rev. 27, 233–246.

European Central Bank (2007) Measuring investors' risk appetite. Financial Stability Review, June: 166–171.

Frye, C., Rhodes, M., Rosellini, R. Svare, B. 2002. The nucleus accumbens as a site of action for rewarding properties of testosterone and its 5alpha-reduced metabolites. Pharmacol. Biochem. Behav. 74, 119–127.

Fuxjager MJ, et al. (2010) Winning territorial disputes selectively enhances androgen sensitivity in neural pathways related to motivation and social aggression. P NATL ACAD SCI USA 107, 12393-12398.

Gai P, Vause N (2006) Measuring investors' risk appetite. Int J Cent Bank. 2, 167–188.

Geniole, S.N., Bird, B.M., Ruddick, E.L. and Carré, J.M., 2016. Effects of competition outcome on testosterone concentrations in humans: an updated meta-analysis. *Hormones and Behavior*.

Guiso L, Sapienza P, Zingales L, 2013. Time varying risk aversion. NBER Working Paper No. 19284 (National Bureau of Economic Research, Cambridge, MA).

Hennessy, J.W. Levine, S, 1979. Stress, Arousal, and The Pituitary-Adrenal System: A Psychoendocrine Hypothesis. Progress in Psychobiology And Physiological Psychology, 8, 133-178.

Hermans, E., Putman, P., Baas, J., Koppeschaar, H. van Honk, J. 2006. A single administration of testosterone reduces fear-potentiated startle in humans. Biol. Psychiat. 59, 872–874.

Hsu Y & Wolf L, 2001. The winner and loser effect: what fighting behaviours are influenced? Animal Behav 61, 777-786.

Kademian SM, Bignante AE, Lardone P, McEwen BS, Volosin M, 2005. Biphasic effects of adrenal steroids on learned helplessness behavior induced by inescapable shock. Neuropsychopharmacol 30, 58–66.

Kandasamy, N., et al., 2014. Cortisol shifts financial risk preferences. P NATL ACAD SCI USA, 111, 3608-3613.

Korte, S. 2001 Corticosteroids in relation to fear, anxiety and psychopathology. Neurosci. Biobehav. Rev. 25, 117–142.

Lehner SR, Rutte C, Taborsky M, 2011. Rats Benefit from Winner and Loser Effects. Ethology 117, 949-960.

Liston C, McEwen BS, Casey BJ, 2009. Psychosocial stress reversibly disrupts prefrontal processing and attentional control. Proc Natl Acad Sci USA 106, 912–917.

Luigino B, Sugden R, 2007. The road not taken: How psychology was removed from economics, and how it might be brought back. Econ J 117, 146–173.

Lupien SJ, et al. 2002. The modulatory effects of corticosteroids on cognition: Studies in young human populations. Psychoneuroendocrinol 27, 401–416.

Määttänen II, Jokela M, Hintsa T, Firtser S, Kähönen M, Jula A, Raitakari OT, Keltikangas-Järvinen L. 2013. Testosterone and temperament traits in men: Longitudinal analysis. *Psychoneuroendocrinol.* 38, 2243-8.

Mah PM, et al. 2004. Weight-related dosing, timing and monitoring hydrocortisone replacement therapy in patients with adrenal insufficiency. Clin Endocrinol (Oxf) 61, 367–375.

Marler, C. Moore, M, 1988. Evolutionary costs of aggression revealed by testosterone manipulations in free-living male lizards. Behav Ecol Sociobiol. 23, 21-26.

Mazur A, Booth A, Dabbs J, 1992. Testosterone and Chess Competition. Soc Psychol Qrtly 55, 70-77.

McEwen BS, et al. 1997. The role of adrenocorticoids as modulators of immune function in health and disease: Neural, endocrine and immune interactions. Brain Res Brain Res Rev 23, 79–133.

McEwen, B. 1998. Stress, adaptation, and disease: allostasis and allostatic load. Ann. N Y Acad. Sci. 840, 33–44.

Mehta, P. H., Snyder, N. A., Knight, E. L., & Lassetter, B. (2015). Close versus decisive victory moderates the effect of testosterone change on competitive decisions and task enjoyment. Adaptive Human Behavior and Physiology, 1(3), 291-311.

Oliveira, R.F., A. Silva, and A.V. Canário, 2009. Why do winners keep winning? Androgen mediation of winner but not loser effects in cichlid fish. Proceed Roy Soc B: Bio Sci. 276, 2249-2256.

Oyegbile TO, Marler CA, 2005. Winning fights elevates testosterone levels in California mice and enhances future ability to win fights. Horm behav 48, 259-267.

Page, L., Coates, J. Winner and loser effects in human competitions. Evidence from equally matched tennis players. Under submission.

Piazza PV, et al., 1993. Corticosterone in the range of stress-induced levels possesses reinforcing properties: Implications for sensation-seeking behaviors. Proc Natl Acad Sci USA 90, 11738–11742.

Piazza, P. V. Le Moal, M. 1997. Glucocorticoids as biological substrate of reward: physiological and pathophysiological implications. Brain Res. Rev. 25, 259–372.

Pope, H.G., Kouri, E Hudson, J, 2000. Effects Of Supraphysiologic Doses Of Testosterone On Mood And Aggression In Normal Men: A Randomized Controlled Trial. Arch Gen Psych, 57, 133-140.

Putman P, Antypa N, Crysovergi P, van der Does WA, 2010. Exogenous cortisol acutely influences motivated decision making in healthy young men. Psychopharm (Berl) 208, 257–263.

Reavis, R. Overman, W. 2000. Adult sex differences on a decision-making task previously shown to depend on the orbital prefrontal cortex. Behav. Neurosci. 115, 196–206.

Rutte C, Taborsky M, Brinkhof M, 2006. What sets the odds of winning and losing? Trends Ecol Evol 21, 16-21.

Salvador, A, Suay, F, Gonzalez-Bono, Serrano, MA, 2015. Anticipatory cortisol, testosterone and psychological responses to judo competition in young men. Psychoneuroendocrinol 28, 364-375.

Sapolsky RM (2000) Glucocorticoids and hippocampal atrophy in neuropsychiatric disorders. Arch Gen Psych 57, 925–935.

Sapolsky, R, 2004. Why Zebras Don't Get Ulcers 3rd ed. New York: Henry Holt.

Sarnyai, Z., McKittrick, C. R., McEwen, B. Kreek, M. J. 1998. Selective regulation of dopamine transporter binding in the shell of the nucleus accumbens by adrenalectomy and corticosterone replacement. Synapse 30, 334–337.

Schroeder, J. Packard, M. 2000. Role of dopamine receptor subtypes in the acquisition of a testosterone conditioned place preference in rats. Neurosci. Lett. 282, 17–20.

Schulz P, Kirschbaum C, Prüßner J, Hellhammer D, 1998. Increased free cortisol secretion after awakening in chronically stressed individuals due to work overload. Stress Med 14, 91–97.

Schwabe L, Wolf OT, 2009. Stress prompts habit behavior in humans. J Neurosci 29, 7191–7198.

Sci Bytes, Where does Science take place? Nature Aug, 29, 2013

Smith, D.R. Whitelaw, R, 2009. Time-varying risk aversion and the risk-return relation. NYU Stern School of Business Working Paper.

Sornette, D. and Cauwels, P, 2015. Financial Bubbles: Mechanisms and Diagnostics, Review of Behavioral Economics: Vol. 2: No. 3, pp 279-305

Stanton, S., Liening, S. Schultheiss, O. 2011. Testosterone is positively associated with risk-taking in the Iowa Gambling Task. *Horm Behav*, *59*, 252–256

Stigler, G.J. and G.S. Becker, 1977. De gustibus non est disputandum. Am Econ Rev: p. 76-90.

Stroud, L.R., Salovey, P., Epel, E. 2002. Sex differences in stress responses: social rejection versus achievement stress. Biol Psychiat. 52: 318-327.

Trainor BC, Bird IM, Marler C, 2004. Opposing hormonal mechanisms of aggression revealed through short-lived testosterone manipulations and multiple winning experiences. Horm Behav 45, 115-121.

van Honk, J., Schuttera, D. J. L. G., Hermansa, E. J., Putmana, P., Tuitena, A. Koppeschaar, H., 2004. Testosterone shifts the balance between sensitivity for punishment and reward in healthy young women. Psychoneuroendocrinol. 29, 937–943.

Wingfield, J., R. Hegner, and A. Dufty, AM Jr., Ball, G, 1990. The "challenge hypothesis": Theoretical implications for patterns of testosterone secretion, mating systems, and breeding strategies. Am. Nat. 136, 829-846.

Wingfield, J.C., Lynn, S, Soma, K. 2001. Avoiding the 'costs' of testosterone: ecological bases of hormone-behavior interactions. Brain, Behav Evol. 57, 239-251.

Zilioli, S., & Watson, N. V. 2014. Testosterone across successive competitions: Evidence for a 'winner effect'in humans? Psychoneuroendocrinology, 47, 1-9.

Str -