Performance Pay and Stress
An Experimental Study

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Discussion Paper in Economics No 17-5
May 2017
ISSN 0143-4543
Performance Pay and Stress – An Experimental Study

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Abstract

Recent economics literature suggests a link between performance pay and ill health, potentially through the adverse effects of performance pay on stress. This project examines this issue using an experimental design that purges the effects of self-selection into performance pay and identifies the direction of causation from performance pay to stress. Results find that those who are paid for their performance experience higher levels of stress, both in terms of perceived stress and objectively measured cortisol levels, than those who are paid by a minimum performance contract.

Keywords: Performance-related pay, real-effort experiment, stress, cortisol

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Acknowledgements: The financial support for this study by the Scottish Economic Society is gratefully acknowledged and appreciated. We are grateful for helpful comments by participants at the 2016 Scottish Economic Society Conference and seminar participants at the University of Aberdeen and the Université Panthéon-Assas as well as Daniel Powell. Help with z-tree programming from Maria Bigoni is also greatly appreciated. All errors remain with the authors.
Performance Pay and Stress – A Pilot Experimental Study

I. Introduction

There is a growing literature investigating different outcomes associated with various types of work contracts. Within the economics literature, there is a focus on the effects of the worker’s payment method on his or her productivity. Thus, it is long advocated by some economists that pay according to performance is the most efficient of the payment schemes (e.g. Lazear 1986). Research on the incidence of performance pay shows a varying incidence of performance-related pay (PRP) in developed countries depending on the industry or occupation. Although the incidence depends on how one defines performance pay some estimates suggest that it is more than 10-15% of European workers and can be as high as 40% in Scandinavia and the US (Bryson et al. 2013). In the main, the literature on performance pay has focused on the labour market effects of such payment schemes although there are a number of studies that investigate the interrelationships between working conditions and wellbeing measures such as job satisfaction. Green and Heywood (2008) find that job satisfaction is higher among those paid by performance while McCausland, Pouliakas and Theodossiou (2005) find that although this relationship holds for higher paid workers, performance pay is correlated with lower job satisfaction for lower paid workers. In view of the above, one should expect that if performance pay has effects on worker’s well-being then it should also be expected that performance pay would have repercussions on the worker’s health. Indeed, as early as 1776, Adam Smith observed in the Wealth of Nations, Book VII that “Workmen … when they are liberally paid by the piece, are very apt to overwork themselves, and to ruin their health and constitution in a few years”.

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There are several potential pathways through which payment methods associated with performance pay may affect the worker’s health. First, there may be an incentive to ‘work harder, not smarter’ and take more risks at work. This could mean that there will be an increase of injuries at work as (particularly manual) workers attempt to increase productivity by overutilising physical capital or using physical capital in unsafe ways. Indirect evidence of this is found in a paper by Freeman and Kleiner (2005) who note that worker’s compensation insurance\(^1\) premiums decreased after a US shoe manufacturer changed from PRP to salaries. A few other studies have shown similar correlations between PRP (generally in the form of piece rates) and injuries in case studies of individual occupations. Interestingly Bender, Green and Heywood (2012) have found that the relationship is much more general as their study finds a very robust relationship between piece rates and injuries using a large survey of over 30,000 workers across the EU. Furthermore, Artz and Heywood (2015) show that these cross-sectional results are robust to controlling for sorting, matching and individual heterogeneity.

A second pathway suggests a relationship between piece rates and physical health measures. Economic theory suggests that PRP explicitly changes the trade-off between work and leisure, giving a relatively larger return to time spent in work. Thus workers are induced by PRP to shift hours to work and away from nonwork activities. Since nonwork activities include time spent on healthy behaviours (such as exercising, sleep or leisure or shopping and cooking home healthy meals) or on activities to reduce stress, an increase of time spent at work may produce a reduction in activities that provide relaxation and reduce stress. The effects of this change would not manifest themselves on immediate health deterioration (as opposed to having an injury), but their adverse repercussion on health would build up over time. There is some

\(^1\) In the US, workers compensation insurance is required by firms to compensate workers for injuries on the job.
empirical support for this by a recent paper by Bender and Theodossiou (2014) who use the 18 waves of the nationally representative British Household Panel Survey (BHPS) to show that the longer the time spent in jobs with performance pay, the higher the odds of having worse overall health, heart problems, stomach problems, and anxiety/depression\textsuperscript{2}. Data from the General Health Questionnaire (GHQ) suggest that the pathway may be through an increase in stress, as there is a strong correlation between time spent in PRP jobs and reported stress level.

In the medical literature (e.g. Jansen et al. 1995, Gozhenko et al. 2009 and Gleitman et al. 2004), the physiological impact of stress or the ‘fight-or-flight’ response is well understood. In response to stress the immune system redirects white blood cells to areas where injury or infection is most likely, the skin becomes cool and sweaty as blood is drawn away from it toward the heart and muscles which are essential for survival, the mouth becomes dry, and the digestive system slows down. However, when the cause of stress passes, the levels of stress hormones drop and the body's various organ systems return to normal, a state called ‘Allostasis’. Importantly, an absent or incomplete relaxation response may cause damage as ‘Chronic Stress’ or ‘Low Grade Stress’ prevents physiological arousal from returning fully to normal. Rohleder (2014) reviews the literature on the effects of acute and chronic psychosocial stress in maintaining abnormal levels of activity. The review suggests that although body's natural defences adapt and thus people can overcome episodes of stress, excessive chronic stress which is constant and persists over an extended period of time can be psychologically and physically damaging. Hence, there is a relationship between inflammatory responses to chronic psychosocial stress and long-term development of disease.

\textsuperscript{2} In another paper, Bender and Theodossiou (2017) examine the link between temporary employment contracts and health and find (as with performance pay) that the longer a worker is in a series of temporary jobs the more detrimental are the outcomes on the health status (approximated by subjective and GHQ measures of health).
Physical effects of chronic stress burden the cardiovascular system, the immune system (increases a person's risk of getting an infectious illness), the brain (interferes with memory and learning), the musculoskeletal system (intensifies the chronic pain of arthritis and produces tension-type headaches) and the reproductive system (can cause impotence in men and affects fertility). In view of these repercussions of chronic stress on health and since most of the life of a working person is spent at work, long exposure to PRP if it generates stress should be expected to generate chronic or low grade stress which can potentially induce severe deterioration of the working person’s health.

However, as Eriksson (2012) points out, one of the biggest problems in identifying econometrically any link between work contracts (including payment methods) and health is that there may be endogeneity between two variables of interest – that is, it is not clear if the causation runs from the type of work contract or payment method to increases in stress (or decreases in health) or if poor health tends to drive workers into certain kinds of contracts or payment methods. The argument is that if a correlation is found between working on a PRP contract and low health status, this may be an outcome of the propensity of individuals with low health status who being unable to perform and hold a job in regular contracts are dislocated to inferior PRP contracts. Alternatively, those who choose PRP contracts might be more adept in stressful situations so subjectively care less about their exposure to stress. To disentangle these effects, econometric research either relies on Heckman (1979)-type corrections for endogenous selection or instrumental variables procedures. In both cases, the unbiasedness of the estimates depends crucially on the statistical properties of the identifying restrictions. Typically, the case for any selection of identifying restrictions to control statistically for this endogeneity can be challenged on statistical or theoretical grounds. The issue could be resolved
if workers were randomly distributed in the different payment contacts, but this is typically not an option in real labour markets.

Hence, in view of the severe repercussions of chronic stress on health, the present study investigates the possible link between PRP and stress by circumventing the endogeneity issues through initial randomisation of the subjects. In the experimental economics literature, there seems to be little research on this linkage. To the knowledge of the authors there is no published research that focuses specifically on the ability of PRP payment schemes to induce psychological stress. Only two papers are somewhat related to the focus of this paper. Thus, Dohmen and Falk (2011) design an experiment examining the sorting of more productive players into situations where there are performance-related payments as a result of the game. However, the linkage between stress and PRP is not central to their study as almost a sidelight, the experimental subjects are asked about their stress and exhaustion at the end of the experiment, and those who are in PRP express higher levels of stress and exhaustion at the completion of the experiment.

The second study is Cadsby, Song and Tapon (2016) who design an experimental study where they examine whether there is increased productivity due to PRP. They also ask the experimental subjects about levels of stress and find that the increase in productivity induced by PRP is fully offset by the increased stress of such a payment scheme such that 25 percent of subjects had lower productivity when paid for their performance particularly among the risk averse.

Both the above papers suggest that there is a link between PRP and stress, but both use self-reported Likert scale responses to a question inquiring about the stress felt by the subjects
during the PRP experience and both allow participants to select into payment schemes according to their preferences – that is, subjects are not randomly assigned to the payment scheme. While self-reported stress may be suggestive of the true underlying, physiological change in stress, it is not an objective measure of stress. Thus, the present study randomly allocates subjects to two groups to circumvent selection bias. In one group, subjects are paid a flat rate while subjects in the other group are paid for their performance.

In addition to the randomisation of the groups, the experiment elicits information about stress before and after the experiment. To follow the previous literature outlined above, subjects are asked to subjectively evaluate their stress level using a Likert-scale. However, these subjective responses are perhaps not as objective as economists might like as a measure of stress.

In psychology and more broadly in the medical literature, stress can be measured objectively through assessing the individual’s heart rate, blood pressure, or cortisol levels in blood or saliva. Cortisol is a hormone secreted when people are in stressful situations. In surveys of the literature on the use of cortisol as a physiological indicator of stress by Kirschbaum and Hellhammer (1989) and Nicolson (2008), it is found that cortisol is quickly released into the body when a person experiences stress. In addition, the cortisol appears in saliva, thus negating the need for invasive blood tests (which may themselves potentially cause stress in subjects). Furthermore, the saliva test remains accurate even at room temperature for at least a week and hence facilitates repeated experiments.

In addition to being an important signal of stress to the body, the release of cortisol has real effects on the body. Under normal circumstances, cortisol (as part of the hypothalamic-pituitary-adrenal or HPA axis) helps to regulate the body’s response to stress by generally
suppressing reactions to stress through allostasis, helping the body return to a normal equilibrium (McEwen 2005). However, repeated or chronic stress can cause ‘allostatic load’ (McEwen 1998) which dampens the ability of the body to return to ‘normal’ either by causing stress reactions such as increased blood pressure to continue beyond the direct impact of stress or by suppressing normal (e.g. immune system) responses to stress (McEwen 1998). While these are simple examples and medical research (e.g. Miller et al. 2007) finds that the mechanisms can be quite complex, current medical research suggests a strong link between cortisol (and other HPA-axis hormones) and adverse health outcomes.

Given its importance as both a signal of stress and its role in the body’s response to stress, subjects are asked to provide saliva samples from which can be derived a measure of changes in salivary cortisol level over the experiment. Thus, the experiment can examine both the subjective and objective changes in stress induced by PRP.

II. The Design of the Experiment

The performance task used in this study was to have subjects calculate a variety of mathematics problems by hand and enter the result in a computer. This is similar to the methodology utilised by Dohmen and Falk (2011). These calculations last for ten minutes which is sufficient time for the rise of cortisol levels in the presence of possible stress. Subjects are randomly assigned either being paid by the number of questions answered correctly (the ‘PRP’ group) or being paid a flat fee for answering ten questions correctly (the ‘nonPRP’ group). During the experiment the computer program ‘z-tree’ (Fischbacher 2007) is used in order to record the correct answers and calculate payoffs. The study protocol has been reviewed and approved by the University of Aberdeen, College of Life Sciences & Medicine Ethics Review Board (CERB/2015/5/1198).
Forty subjects (in two sessions of 20 subjects each) were invited to the Scottish Experimental Economics Lab (SEEL) at the University of Aberdeen to generate the data. The students were recruited using the Online Recruitment System for Economic Experiments (ORSEE) database of potential subjects which is maintained by SEEL. Students were given details about the broad parameters of the experiment and the procedure of the cortisol sample testing. The subjects were also advised, in line with standard cortisol testing protocols, to abstain from eating, drinking caffeine, smoking or taking exercise two hours before the commencement of the experiment. To this effect reminders were sent via email 24 hours before the experiment was scheduled to take place. The two experimental sessions took place at 1500 hours on the Monday and the Wednesday of the same week to control for the known diurnal patterning of cortisol production and to standardise the experience of participants. Saliva samples and subjective stress reports were obtained before and after participation in the experiment. These provide the objective and subjective the stress measures for the individual subject, respectively.

The random assignment of PRP to subjects addresses the issues of endogeneity and non-random selection discussed above. Therefore, the experiment tests whether there is a direct causal relationship between PRP and stress level by examining differences in cortisol (and the subjective measures of stress) across the two groups. Thus one should expect that any comparisons should be representative of the direction and the strength of the PRP – stress relationship directly without any interference of the self-selection effects.

The Experiment: Upon arrival to SEEL, subjects were given a consent sheet that they signed and were randomly allocated a seat at a computer terminal. Screens between terminals prevented subjects from seeing other subjects and their terminals. When all 20 subjects were
registered, an outline of the experiment was read to the group and any questions were answered. All subjects were informed that for their participation they would earn £5, with the opportunity to earn more money during the experiment.

In the next stage a baseline of stress measurement for the subjects was obtained. First, instructions were given for the cortisol test, which involved chewing a cotton swab (SalivaBio Oral Swab, Salimetrics Europe) for 60 seconds and then placing the swab in a test-tube. Subsequently, a computer-based questionnaire was given to each subject, with a number of subjective questions about current stress levels, asking the subjects whether they felt stressed (as in Dohmen and Falk3).

After the completion of the questionnaires about stress, all subjects were given a practice round of answering three mathematical questions – one addition (in thousands), one multiplication (hundreds by tens) and one division (thousands by tens). Subjects were allowed to use a scratch piece of paper, but no calculator. This part of activity was not timed. After everyone completed this practice round, the computer program randomly assigned subjects into the PRP or nonPRP group. Subjects were individually told by the computer how they would be paid. The payment schemes were as follows: 20p for each correct answer for those randomly allocated to the PRP group and a £5 flat payment for the nonPRP group if at least ten questions were answered correctly. Subjects worked independently and were not aware of what the alternative payment schedule was or of how other subjects were being paid. Subjects were told that they had ten minutes to do the mathematics questions and that there were a maximum of 50 questions. When all were ready, a clock appeared on all screens counting down the time in seconds. During the task, the number of questions answered correctly was also displayed at the top of

3 The question is: ‘How stressed do you feel today? (1=not stressed at all, 5=very stressed)’. 
the screen. The mathematical questions (either multiplication, addition, or division) were shown in the middle of the screen. When those in the nonPRP group accomplished the minimum performance target of ten correct answers, this was indicated at the top of the screen. Although they were not eligible to receive any additional pay and were told this on their screen, subjects in this group could still continue to answer questions if they wished.

At the end of ten minutes, the task was stopped. Because the cortisol response to an acute stressor peaks around 20 minutes after stressor onset (Kirschbaum, Pirke and Hellhammer 1993 and Dickerson and Kemeny 2004), the subjects were asked to leisurely complete several tasks for the 10 minutes after the (10 minute) experiment was complete. First, they answered another round of the computer based subjective questions about stress and provided some demographic information (e.g. gender, year at university, broad discipline of studies and age) before completing two nonstressful filler tasks (rating vignettes of potential jobs with different characteristics and colouring patterns). At the end of the ten minutes, subjects took a second cortisol test by chewing on another swab for one minute and putting the swab in a different, labelled test-tube after the minute was over.

Finally, subjects were called to the control room by seat number to get their payment and were thanked for their participation. Test-tubes were transferred into a freezer and when both sessions were complete, the frozen samples were sent to a laboratory (Salimetrics, Europe) for analysis of cortisol levels for each subject before and after the task.

III. Results

The individuals in the combined samples average 22.7 years of age and 57.5% are female. The computer generated randomisation allocated 57.5% (23 out of 40) to the PRP group. On
average, PRP subjects answered 34.7 questions correctly (SD 9.6) whereas the nonPRP answered 32.1 questions correctly (SD 11.4). The standard deviation regarding the correctly answered questions is greater for the nonPRP mainly due to the fact that some subjects stopped after meeting the minimum requirement of correct questions. The average pay-out was £11.90 for the PRP group ranging from £8.80 to £14.60 and £10 for the nonPRP group. It is noteworthy that all nonPRP subjects obtained the minimum required performance.

Stress Measurements before and after Treatment: An initial analysis of the cortisol measures of stress identified one subject whose cortisol measurement in the first assay was assessed to have a hormone level more than four standard deviations from the mean level of cortisol for the other 39 subjects. Given the likelihood that this measure reflected contamination and the potential for this clear outlier to affect comparisons, the information for this particular subject was excluded from the subsequent analysis, leaving 39 subjects in the analysis.

Figure 1 shows the means of the stress levels for the cortisol and subjective measurements at the start (before PRP assignment) and the end of the experiment. A clear pattern emerges from this figure with the objective and subjective measures of stress increasing for the PRP group and falling for the nonPRP group. Figure 2 shows the differences between the start and the end of the experiment for the stress of the two groups for the two measures, again showing that for the PRP group there is some positive elevation in stress reflected in both cortisol and subjective measures of stress but there are noticeable decreases for the two measures in the nonPRP group.

Differences in Cortisol. While suggestive, the figures do not indicate whether these changes are statistically different. In order to test this, first there is a need to establish that both the
PRP and the nonPRP groups have non-significant differences in the levels of mean stress before the treatment. To assess the validity of this requirement a t-test comparing the level of stress for the PRP and nonPRP groups before treatment is employed.

Snedecor and Cochran (1989) suggest that unequal variances can influence the analysis. Hence in the first step, the Folded F-test for unequal variances is used to evaluate if equality of variances can be assumed. If there is support for inequality of variances, the Satterthwaite test for unequal variances is utilised, otherwise the Pooled test for equal variances is used. Snedecor and Cochran (1989) suggest that when in doubt it is safer to assume unequal variances.

In view of the above, first the homogeneity of variances between the two groups is assessed using the Folded F-test (see Table 1) that indicate that the two groups may have unequal variances with a p-value of 0.0343. In view of this, the Satterthwaite Unequal Variances test is employed giving a test statistic of 1.90 which shows that the null hypothesis of equality of means cannot be rejected at the 0.05 level of statistical significance.

Therefore, a Two-Sample t-test for the difference of the changes of stress between the start and the end of the experiment for the two groups is employed, under the null that there is no difference in the elevation of stress between the PRP and the non-PRP group. The Folded F-test for homogeneity of variances post-treatment shows that the null cannot be rejected with a p-value of 0.1867. Hence the Pooled for Equal Variances t-test reveals that the differences in the changes of stress between PRP and nonPRP is statistically significant with a p-value of

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4 The statistical analysis below is performed using the software Statistix 10.0.
0.0476. Given that the stress level of the PRP group went up, then this lends support to the conjecture that the PRP payment scheme is inducing stress in subjects.

**Differences in Subjective Stress.** The above statistical procedure is also used for the subjective stress measure. The Folded F-test for the equal variances before assignment suggest that there is no difference in variances in the subjective stress measure (p-value of 0.3327). Thus, a standard t-test of equal means across the two groups cannot reject the null of equal means of the subjectively measured stress at the start of the experiment (p-value of 0.2068).

For the post-treatment differences in variances, a Folded F-test suggests no difference in variance (p-value of 0.3567). Interestingly, however, the Pooled for Equal Variances test reveals that the differences in the elevation of subjective stress between PRP and nonPRP are statistically insignificant (p-value of 0.2897). Why this is so, given the statistically significant difference in the change in the cortisol measurement is not quite clear. It may be that subjective and objective stress are measuring different things, or that respondents are not aware of the body’s response to stress. It is also possible, given the limited values of the subjective stress response (one to five) there may have been the inability to report higher levels of stress because of the index used. Future replications of the study will address this by given a larger range in the responses to this variable.

**Productivity Measurement:** Figure 3 shows that differences in productivity between the PRP and the nonPRP group as reflected in the group mean in correct answers during the experiment. The figure suggests no great difference in productivity between the two groups. This is confirmed by the formal statistical test. The Folded F-test cannot reject (p-value of 0.224) the null of equal variances. Testing the equality of the average number of correct answers, the
Pooled for Equal Variances t-test shows that the difference in productivity between the two groups is statistically insignificant (p-value: 0.4515).

IV. Conclusions

This study provides a real-effort experiment to reveal whether there is a link between PRP and stress. The experiment uses random assignment to PRP to circumvent concerns of self-selection and endogeneity bias and measures stress by both subjective and objective means. Results suggest that the subjective measure of self-reported stress levels and the objective stress measure obtained by measuring cortisol move in a similar direction for the PRP and nonPRP groups, but only the movement in the cortisol shows statistically significant differences between the two groups. This might suggest that individuals are underestimating their stress at the end of the experiment, though more work will be needed to test this formally.

Although the results are suggestive that PRP can generate stress, this study is a small pilot and is based only 39 respondents. Future work will expand on this sample to measure the effects of PRP on stress more accurately. Some tweaks in the experimental design are also likely needed. For example, expanding the range of answers for the subjective stress will give more degrees of freedom for stress to change. In addition, more needs to be done to relax subjects when they enter the lab given the relatively high levels of stress at the beginning of the experiment.

In addition to these extensions, other variations on the experiment might yield further interesting comparisons. For example, while the above design allows for random assignment of PRP (perhaps similar to a ‘real world’ change in the employment contract for a firm), many employment situations involve the element of choice of PRP or nonPRP (e.g. when people are
looking for jobs) and the interaction between stress and PRP may be different in these cases. Allowing subjects to choose a PRP or nonPRP job would allow for this selection in an experimental setting. A ‘cross-over’ design, where in a third round some of the PRP subjects move to the nonPRP group while some nonPRP subjects move to the PRP group, would also add a further dimension to the analysis.
Figure 1. Cortisol and Subjective Stress Measurements; Before and After Treatment

Cortisol Measurement

Subjective Stress
Figure 2: Cortisol and Subjective Measurements; Before and After Treatment

Cortisol Measurement

-0.05
-0.03
-0.01
0
0.01
PRP - Difference
NonPRP - Difference

Subjective

0.35
0.3
0.25
0.2
0.15
0.1
0.05
0
0.05
PRP Difference
NonPRP Difference


Figure 3. Productivity Measurement; Correct Answers.
<table>
<thead>
<tr>
<th>Test</th>
<th>Type of test</th>
<th>Cortisol Measurement</th>
<th></th>
<th>Subjective Stress Measurement</th>
<th></th>
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<td>Equal variances before assignment</td>
<td>Folded F-test</td>
<td>16, 21</td>
<td>2.35 (0.0343)</td>
<td>16, 21</td>
<td>1.21 (0.3327)</td>
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<tr>
<td>Equality of differences in change in stress</td>
<td>Satterthwaite Unequal Variances test</td>
<td>26.1</td>
<td>1.90 (0.0679)</td>
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<tr>
<td>Equality of means before assignment</td>
<td>t-test for mean difference</td>
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<td>-1.28 (0.2068)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Equal variances after treatment</td>
<td>Folded F-test</td>
<td>21,16</td>
<td>1.55 (0.1867)</td>
<td>21,16</td>
<td>1.20 (0.3567)</td>
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<tr>
<td>Equality of differences in change in stress</td>
<td>Pooled for Equal Variances t-test</td>
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<td>2.11 (0.0476)</td>
<td>37</td>
<td>1.07 (0.2897)</td>
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References:


