Complementary Therapies in Medicine 37 (2018) 103–109

Contents lists available at ScienceDirect

Complementary Therapies in Medicine

journal homepage: www.elsevier.com/locate/ctim

A comparison of the cardiovascular effects of simulated and spontaneous laughter

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ARTICLE INFO

Keywords:
Laughter
Heart rate
Heart rate variability
Psychophysiology
Simulated
Spontaneous

ABSTRACT

Objectives: Laughter has long been regarded as beneficial for health, but the mechanisms are not clearly understood. The current study aimed to compare the acute cardiovascular effects of spontaneous and simulated laughter.

Design: A mixed factorial experiment was performed to examine changes in cardiovascular variables in response to experimental tasks across conditions.

Interventions: A sample of 72 participants were randomised to one of three 6 min interventions. Participants in the simulated laughter condition were asked to generate fake laughter, the spontaneous laughter condition viewed a humorous video, and the control condition watched a non-humorous documentary. This was followed by a laboratory stress task.

Main outcomes measures: Heart rate and heart rate variability (as indexed by rMSSD) were monitored continuously throughout the experiment using ECG.

Results: The simulated laughter condition had a significantly higher heart rate ($p < .001$, $\eta_p^2 = .26$) and lower rMSSD ($p < .001$, $\eta_p^2 = .13$) during the laughter task compared to the other two conditions. Follow-up hierarchical regressions indicated that the difference in heart rate was due to the fact that the simulated condition produced more laughter. The difference in rMSSD, however, was unique to the simulated condition even when controlling for the amount of laughter. The simulated laughter condition had a significantly lower mean HR during the stress task but this was not significant after controlling amount of laughter produced.

Conclusions: Laughter leads to increased heart rate and reduced heart rate variability, which is similar to the effects of exercise. This finding is more pronounced in simulated laughter.

1. Introduction

In popular culture, laughter is perceived as being beneficial for one's health, and some research supports this. Different forms of laughter have been found to improve mood,[1] reduce depression,[2] improve immune function,[3] decrease pain,[4] and reduce stress hormone concentrations.[5,6] However, other studies have shown that laughter may be detrimental to people with particular health conditions in the short-term, including asthmatics.[7] and those with chronic obstructive pulmonary disease[8]

Research has focussed on two main types of laughter; simulated and spontaneous laughter.[9] Spontaneous laughter, commonly referred to as 'real' laughter, is triggered by external humorous stimuli, and occurs in the presence of positive emotions. Spontaneous laughter is unique as it elicits involuntary contractions of the orbicularis oculi muscles in the eye socket, a phenomena known as the Duchenne Smile.[10] In contrast, simulated laughter is triggered by oneself at will and therefore is not elicited by humorous stimuli or positive emotions.[9] This form of laughter is commonly known as 'fake' or voluntary laughter and involves laughing on command. Simulated laughter can be performed by any individual using controlled vocal sounds (e.g. ha, he, ho) and is gaining popularity as a form of therapy.[11] For example, the Laughing Qigong Programme uses a combination of simulated laughter and qigong techniques as a standardised therapy which has been shown to improve mood and decrease stress markers.[12]

It has been theorised that laughter is beneficial because it is a form of aerobic exercise. Like exercise, laughter is stimulating: it increases heart rate and blood pressure, enhances immune functioning and exercises skeletal muscle.[13] Laughter activates internal oblique muscles to similar levels as crunches and back lifting exercises.[14] Laughter also consistently causes changes in respiration levels similar to exercise: lung volume decreases, respiration rate increases, and compression is

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https://doi.org/10.1016/j.ctim.2018.02.005
Received 11 January 2018; Received in revised form 1 February 2018; Accepted 15 February 2018
Available online 21 February 2018

applied to the airways.[15] The parallels drawn between laughter and exercise demonstrate a possibility that the health effects of laughter are merely due to exercise effects, rather than from anything unique to laughter. Therefore, the act of laughter itself is the critical component, even in the absence of humour.[9] This mechanism implies there is little need to distinguish between spontaneous and simulated laughter as both should produce the same physiological effects on the body.

A related theory is the Motion Creates Emotion Theory. Dr. Kataria, the founder of laughter yoga, argues that both simulated and spontaneous laughter can lead to the same physiological and psychological health benefits.[16] This theory states that while the human mind can tell the difference between simulated and spontaneous laughter, the human body cannot.[10] However, simulated and spontaneous laughter have never been compared within the same study.

An important indicator of how well the body responds to a stimulus, and in particular exercise, is heart rate (HR) and heart rate variability (HRV). HRV is the normal rhythmic variations in consecutive heart beats that index the cardiovascular system's ability to meet demands.[17] HRV represents both the sympathetic and parasympathetic effects on the heart. Stress or exercise triggers parasympathetic withdrawal and subsequently sympathetic activation. This leads to increased HR and decreased HRV as the underlying dynamic switches from inhibitory to excitatory dominance to allow the system to meet the challenge at hand.

The effects of laughter on the cardiovascular system have been investigated in only a few studies to date. Laughter produced by watching a humorous video increased sympathetic nervous system arousal as indexed by increased galvanic skin resistance, increased heart rate (HR) and decreased finger temperature.[18] Similarly, watching a humorous video produced a significant increase in HR and blood pressure, compared to watching a control video.[19] These studies demonstrate that laughter can produce changes in cardiovascular function.

To test the theory that both simulated and spontaneous laughter are forms of exercise that can stimulate the cardiovascular system similarly, this study aimed to compare the acute cardiovascular effects of simulated and spontaneous laughter. The study also compared the ability of these two types of laughter to buffer the cardiovascular stress response to a laboratory stress task. Lastly, the study investigated whether the effects observed were correlated with the amount of laughter produced. This final aim was included as past research on laughter has failed to actually correlate laughter with health outcomes.

It was hypothesised that simulated and spontaneous laughter would have similar cardiovascular effects (increased HR and decreased HRV) during the laughter task, and these cardiovascular responses would be significantly larger than the control condition. It was expected that the simulated and spontaneous laughter conditions would exhibit similar cardiovascular responses to the stress task but smaller cardiovascular responses (decreased HR and increased HRV) compared to the control condition. It was further hypothesised that the amount of laughter produced during the laughter task would predict the cardiovascular outcomes above and beyond the effect of condition and adding condition as a predictor would not significantly increase the amount of variance explained.

2. Method

2.1. Design

A 7 (task) × 3 (condition) mixed factorial experiment was performed to examine the acute changes in cardiovascular variables overtime in response to experimental tasks across conditions (spontaneous laughter vs. simulated laughter vs. control).

2.2. Sample

A sample of 72 adults (48 female, 24 male; average age 24.15 years, SE = 1.00) was recruited from advertisements to the general public and University students. Inclusion criteria were those aged 18–64 who could give informed consent. The exclusion criteria included: people with cardiovascular conditions, those taking regular medication which may affect the cardiovascular system, women who were pregnant, those with asthma and those with clinical depression or anxiety. Participants were randomised to one of the three conditions prior to the experimental setting on a 1:1:1 basis. The experimenter was not blinded to condition allocation.

Ethics Approval was granted by the University of Auckland Human Participants' Ethics Committee, and participant written informed consent was obtained.

2.3. Cardiovascular measures

The primary outcome was cardiovascular functioning as measured by heart rate (HR, bpm) and heart rate variability (HRV). HR and HRV were continually measured throughout the experiment using a standard 3-lead ECG attached to the participants chest. Data was collected using Mindware Bio lab 3.02 software with a 1000 Hz sampling rate and was analysed using Kubios HRV version 2.2 software. Root Mean Square of the Successive Differences (rMSSD) was used as an index of vagally-mediated HRV, which was calculated by analysing the intervals between the R-Spikes during each time period. rMSSD reflects the parasympathetic activity on the heart, as opposed to an overall measure of HRV.

2.4. Laughter intensity and frequency scale (LIFS)

The amount of laughter produced by each participant was observationally coded to check whether the changes in the cardiovascular variables were correlated with the actual occurrence of laughter as this has been a limitation in past laughter research. A systematic coding schedule was designed, the Laughter Intensity and Frequency Scale (LIFS), adapted from Bennett's[20] Humour Response Scale, with more clearly operationalised definitions of laughter[21] Each participant was rated by the lead researcher on two separate scales for intensity (0 = no laughter to 3 = nearly continuous laughter) and frequency (0 = no laughter to 3 = belly laughter) at the end of each one minute period. Scores for each minute were totalled for each scale to get an overall intensity and frequency scale out of 18. These scores for each scale were then combined to give an overall score for the six minutes ranging from 0 to 36. The full scale is provided in the Appendix A.

2.5. Procedure

All participants were asked to avoid exercise, alcohol, tobacco and caffeine the 24 h before the experiment and to refrain from eating and drinking two hours prior. Participants were seated in front of a computer which prompted the experimental tasks. The procedure is shown in Fig. 1. During the resting periods, participants were asked to sit as still as possible and not to move, talk or close their eyes, in order to measure resting cardiovascular activity.

During the intervention period, participants were asked to perform a task, specific to their allocated condition which lasted six minutes. The participant's responses to the task were video-recorded for later analysis. Those in the simulated laughter condition were instructed to generate as much simulated laughter as they could for six minutes. Participants in the spontaneous laughter condition viewed a six minute stand-up comedy routine on video. Lastly, participants in the control condition viewed a six minute documentary on Paua farming which was deemed by the researcher to be interesting, yet emotionally neutral.

After their assigned intervention, participants were exposed to a shortened version of the Trier Social Stress Test (TSST).[22] Participants were given three minutes to prepare and three minutes to present a speech to convince the experimenter to give them their dream job.

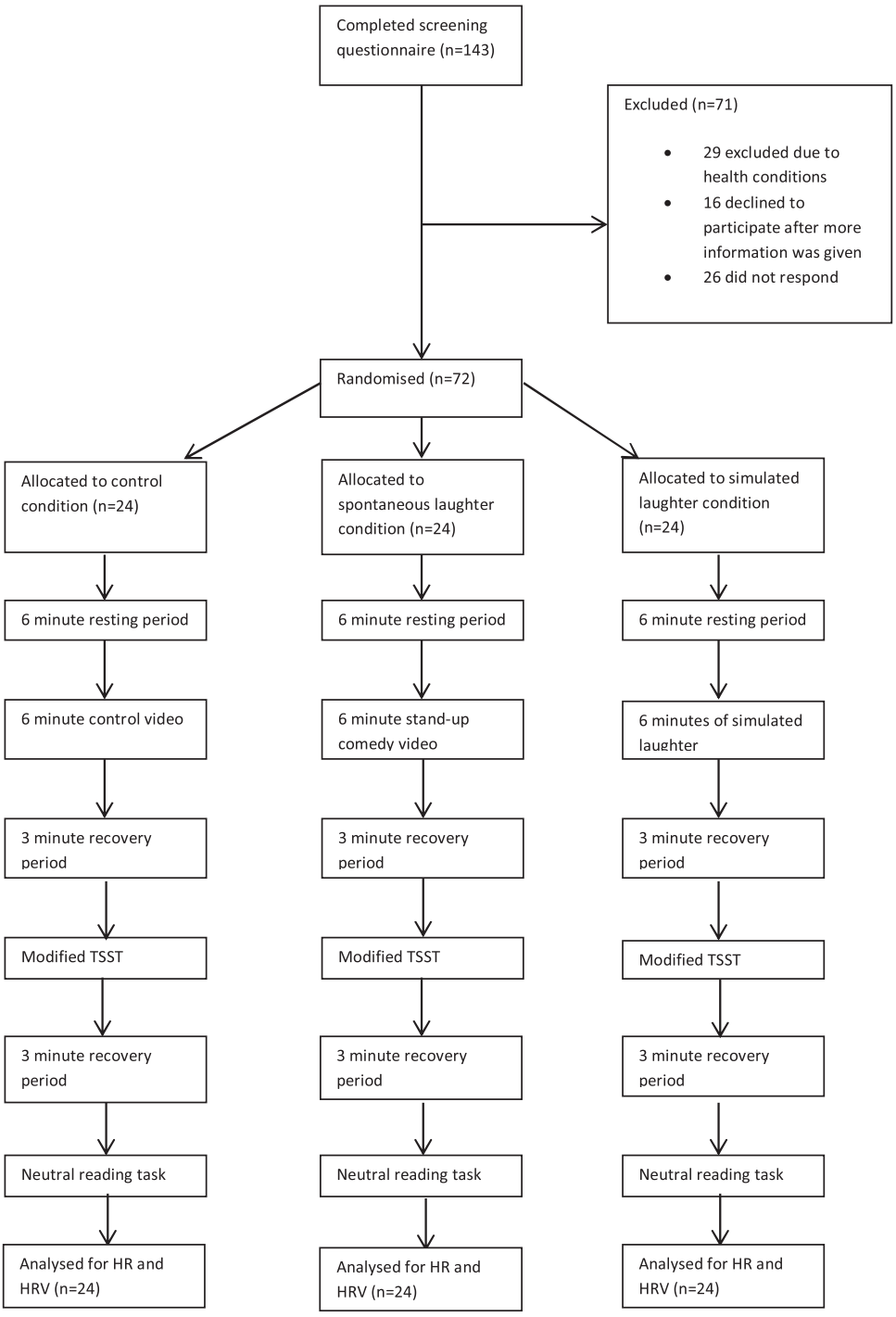

Fig. 1. Flow chart showing the procedure of the study.

Participants were told their speech would be recorded and that a panel of judges would review it and award the best speech with a $100 voucher.

Lastly, participants read an affectively neutral document (a set of washing instructions) out-loud for three minutes as a manipulation check to ensure the physiological and psychological responses caused by the TSST were due to the stressful nature of the task as opposed to just the effect of speaking.[23]

2.6. Statistical analysis

Data were analysed using IBM SPSS Statistics 22. Mixed factorial ANOVAs were completed to analyse the interaction and main effects of task (baseline, laughter task, laughter recovery, speech preparation, speech presentation and reading) and condition (simulated, spontaneous and control) on HR and rMSSD.

rMSSD violated the assumption of normality for ANOVAs and therefore was transformed using a natural log transformation and logged values were used in analyses. All tests were reported using the Greenhouse-Geisser adjustment due to violations in sphericity.[24] All significant interaction effects were followed up using simple pairwise comparisons with bonferroni corrections.

A 3-step hierarchical regression was conducted for all significant interactions to determine the amount of variance predicted by both the

Table 1
Summary of Demographic and Baseline Characteristics of Participants across Condition.

Baseline variable	Simulated	Spontaneous	Control	p-value
Age (years) M(SE)	27.25(2.21)	21.54(0.71)	23.67(1.77)	.060[a]
Gender (%)				.472[b]
Female	16(67%)	14(58%)	18(75%)	
Male	8(33%)	10(42%)	6(25%)	
Ethnicity (%)				.118[b]
NZ European/Pakeha	10(14%)	6(8%)	13(18%)	
Non-European/Other	14(20%)	18(25%)	11(15%)	
BMI M(SE)	25.15(1.33)	22.83(0.80)	23.71(0.71)	.251[a]
Alcohol (drinks/week)M (SE)	3.17(0.80)	0.71(0.34)	2.17(0.53)	.016[a]
Caffeine (cups/day) M(SE)	1.70(0.42)	0.79(0.23)	1.00(0.26)	.108[a]
Sleep (hours/night) M(SE)	7.13(0.16)	7.42(0.20)	7.79(0.19)	.045[a]
Base HR (bpm) M(SE)	73.75(2.51)	77.78(2.39)	78.10(2.31)	.383[a]
Base rMSSD (ms) M(SE)	37.69(4.15)	42.32(6.14)	43.75(4.38)	.773[a]

Note: M = Mean, SE = Standard error, % = percentage of participants in that category. P-value was calculated by one-way ANOVAs[a] and Chi-square tests[b].

amount and type of laughter. In the first step of the model, covariates known to affect the cardiovascular system were included, including BMI, alcohol, age, gender and exercise. The second step included the LIFS scores to determine the variance in the outcome predicted by the amount of laughter produced. In the third step, condition was included to determine if the type of laughter task predicted any further variance on top of the amount of laughter. Due to the categorical nature of the conditions, condition was dummy coded before being added into the analysis. A p value of .05 was maintained.

3. Results

3.1. Baseline characteristics

A shown in Table 1, significant group differences at baseline were found for sleep, ($F_{(2,68)} = 3.24$, $p = .045$) and alcohol consumption ($F_{(2,69)} = 4.38$, $p = .016$). The group differences in the average amount of alcohol consumed per week was moderate to large and previous research has found that alcohol can have a large effect on cardiovascular reactivity.[25] Therefore, alcohol consumption was included as a covariate in all remaining analyses. The analyses were repeated with sleep as an additional covariate, but this made no difference to the significance of the results, and for reasons of simplicity, analyses are reported without sleep as a covariate.

3.2. Manipulation checks

A one-way ANOVA was conducted to examine whether scores on the LIFS differed across conditions. Significant group differences were found for LIFS score ($F_{(2,69)} = 139.30$, $p < .001$). The control condition ($M = 0.13$, $SE = 0.61$) had significantly lower mean LIFS score than both the simulated ($M = 28.21$, $SE = 1.29$, $p < .001$) and spontaneous conditions ($M = 12.00$, $SE = 1.61$, $p < .001$). The simulated condition had a significantly higher mean LIFS score than the spontaneous condition ($p < .001$).

As another manipulation check, a series of simple Pearson's correlations were conducted in order to check whether changes in the cardiovascular variables were correlated with LIFS scores, irrespective of condition. Both mean HR ($r = .48$, $p < .001$) and rMSSD ($r = -.38$, $p = .001$) during the laughter task were significantly correlated with LIFS scores.

3.3. Differences across conditions

A series of 3(condition) × 7(task) mixed factorial ANCOVAs were conducted to evaluate the effects of condition and task on HR and lnrMSSD whilst controlling for the effect of alcohol. No significant main effects were observed for the effect of condition on HR ($F_{(2,68)} = 0.39$, $p = .677$, $\eta_p^2 = .01$) or lnrMSSD ($F_{(2,68)} = 1.23$, $p = .30$, $\eta_p^2 = .03$). Significant main effects of task were found for HR ($F^{3,200)} = 56.90$, $p < .001$, $\eta_p^2 = .46$) and lnrMSSD ($F_{(4,261)} = 10.24$, $p < .001$, $\eta_p^2 = .13$). Means, standard errors and post-hoc comparisons between the tasks for each cardiovascular variable are provided in Table 2.

As shown in Fig. 2, there was a significant interaction effect on HR while controlling for the effects of alcohol ($F_{(6,200)} = 15.74$, $p < .001$, $\eta_p^2 = .32$). Follow-up comparisons indicated significant differences between conditions during the laughter task ($F_{(2,68)} = 11.85$, $p < .001$, $\eta_p^2 = .26$). The simulated condition ($M = 91.14$, $SE = 2.47$) had a significantly higher HR during the laughter task than both the spontaneous ($M = 77.73$, $SE = 2.50$, $p = .001$) and control conditions ($M = 75.29$, $SE = 2.41$, $p < .001$). The spontaneous and control conditions did not significantly differ from each other in mean HR during the laughter task.

The conditions also significantly differed in mean HR during speech presentation ($F_{(2,68)} = 4.52$, $p = .0.14$, $\eta_p^2 = .12$). The simulated laughter condition had a significantly lower mean HR during the speech presentation ($M = 85.70$, $SE = 2.77$) than both the spontaneous ($M = 96.75$, $SE = 2.79$, $p = .024$) and control conditions ($M = 95.26$, $SE = 2.70$, $p = .046$). The spontaneous and control conditions did not significantly differ from each other. The other tasks did not show any significant differences in mean HR across conditions.

As can be seen in Fig. 3, a significant interaction effect for task and condition on lnrMSSD was observed ($F_{(8,261)} = 5.02$, $p < .001$, $\eta_p^2 = .13$). Follow-up ANCOVAs demonstrated that the conditions differed significantly in lnrMSSD during the laughter task only ($F_{(2,68)} = 9.61$, $p < .001$, $\eta_p^2 = .22$), with the simulated condition having significantly lower mean lnrMSSD during the laughter task ($M = 3.02$, $SE = 0.12$) than both the spontaneous ($M = 3.57$, $SE = 0.12$, $p = .005$) and control conditions ($M = 3.70$, $SE = 0.11$, $p < .001$). The spontaneous and control conditions did not differ ($p > .05$).

Table 2
Summary Statistics and Post-hoc Comparisons for Cardiovascular Variables across Tasks Averaged across Groups.

Task		Baseline	Laughter task	Laughter recovery	Speech preparation	Speech presentation	Speech recovery	Reading
Cardiovascular Variables	HR(bpm) M (SE)	76.76(1.37) b**d**e**g**	81.39(1.39) a**c**d*e**f**	75.63(1.25) b**d**e**g**	86.19(1.48) a**b*c**e**f**g**	92.57(1.56) a**b**c**d**f**g**	75.21(1.20) b**d**e**g**	79.67(1.16) a**c**d**e**f**
	rMSSD(ms) M(SE)	39.88(2.73) d*e**f*	36.67(2.60) c*e*f**	41.57(3.07) b*d*e**	33.55(2.31) a*c*f**g*	28.94(1.75) a**b*c**f**g**	44.92(3.43) a*b*d**e**g*	36.86(1.95) d*e**f*
	lnrMSSD M (SE)	3.53(0.07) d*e**f*	3.43(0.07) c*e*f**	3.57(0.07) b*d*e**	3.37(0.06) a*c*f**g*	3.24(0.06) a**b*c**f**g**	3.64(0.07) a*b*d**e**g*	3.51(0.05) d*e**f*

Note: a = different to baseline, b = different to laughter task, c = different to laughter recovery, d = different to speech preparation, e = different to speech presentation, f = different to speech recovery, g = different to reading, *p < .05, **p < .001
Note: Values for rMSSD variables have been reported as both absolute and log values. This is due to the fact that rMSSD was positively skewed and thus have been logged transformed for analysis.

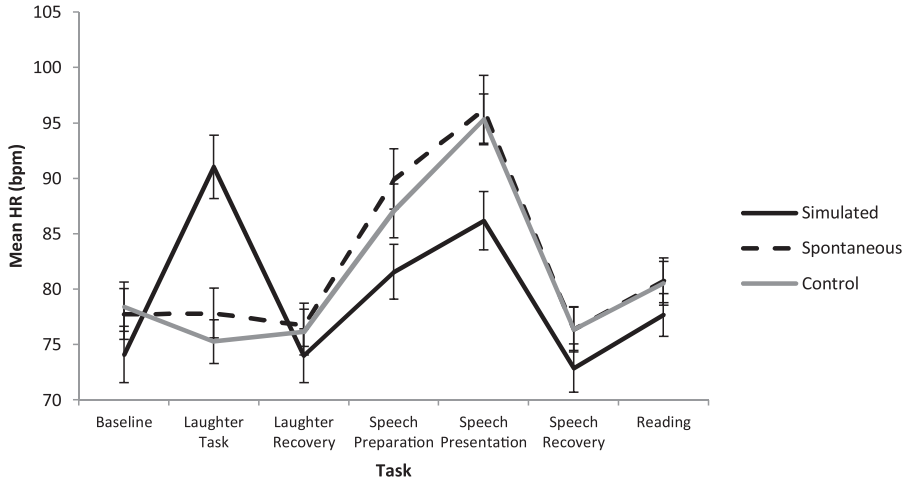

Fig. 2. Mean HR across tasks split by condition.

3.4. Regressions

A hierarchical regression assessed the effect of LIFS score and condition on mean HR during the laughter task. Step 1 of the regression model (containing the known covariates of BMI, alcohol, age, gender and exercise) was not significant ($F_{(5,66)} = 2.13$, $p = .073$, $R^2 = .14$). Step 2 (with the addition of LIFS score) was significant ($F_{(6,65)} = 6.61$, $p < .001$, $R^2 = .38$, $\Delta R2 = .24$) explaining 38% of the variance in mean HR during the laughter task. LIFS score was a significant predictor when controlling for known covariates ($b = 0.53$, $\beta = .51$, $t.^{65} = 5.01, p < .001$). This indicates that a higher LIFS score predicted higher HR during the laughter task. Step 3 of the model (with the addition of condition) was significant ($F_{(8,63)} = 6.56, p < .001, R^2 = .45$) explaining 45% of the variance in mean HR during the laughter task. However, neither dummy variable was found to significantly predict any variance in mean HR during the laughter task when controlling for the known covariates and LIFS scores ($ps > .05$). This indicates that condition did not significantly explain any further variance in HR when controlling for the amount of laughter produced.

Another hierarchical regression analysed the effects of LIFS score and condition on mean HR during speech presentation. Step 1 (containing known covariates) was significant ($F_{(5,66)} = 3.22$, $p = .012$, $R^2 = .20$) indicating that the covariates predicted 20% of the variance in mean HR. Step 2 (with the addition of LIFS score) was also significant ($F_{(6,65)} = 3.26$, $p = .007$, $R^2 = .23$) explaining 23% of the variance in mean HR during speech presentation. However, LIFS score was not a significant predictor when controlling for the covariates ($b = -0.21$, $\beta = -.19$, $t^{65} = -1.72$, $p = .090$). Therefore, the amount of laughter produced by each participant was not a significant predictor of mean HR during speech presentation. Step 3 (with condition) was significant ($F_{(8,63)} = 2.72$, $p = .012$, $R^2 = .26$) explaining 26% of the variance in mean HR during speech presentation. However, condition did not significantly predict any variance in mean HR during speech presentation when controlling for known covariates and LIFS scores ($ps < .05$).

A third hierarchical regression was conducted to examine the effects of LIFS score and condition on lnrMSSD during the laughter task. Step 1 of the model (containing known covariates) was not significant ($F_{(5,66)} = 1.56$, $p = .183$, $R^2 = .11$). Step 2 (with the addition of LIFS score) was significant ($F_{(6,65)} = 3.64$, $p = .004$, $R^2 = .25$) explaining 25% of the variance in lnrMSSD during the laughter task. LIFS score was a significant predictor of lnrMSSD when controlling for the effects of the covariates ($b = -0.02$, $\beta = -.40$, $t^{65} = -3.56$, $p = .001$). A 1 point increase in LIFS score predicts a 0.02 unit decrease in lnrMSSD. Adding LIFS score to the regression model explained a further 18% of variance in lnrMSSD during the laughter task ($\Delta R^2 = .18$). This finding indicates that the amount of laughter produced in the laughter task predicted a decrease in rMSSD. Step 3 (with the addition of condition) was significant ($F_{(8,63)} = 34.00$, $p = .001$, $R^2 = .34$) explaining 34% of the variance in lnrMSSD during the laughter task. The simulated dummy variable was found to be a significant predictor when controlling for the covariates and LIFS score ($b = -0.80$, $\beta = -.61$, $t^{63} = -2.29$, $p = .026$). The simulated condition had 0.80 units of lnrMSSD lower than the control condition during the laughter task when controlling for covariates and LIFS scores. The spontaneous group

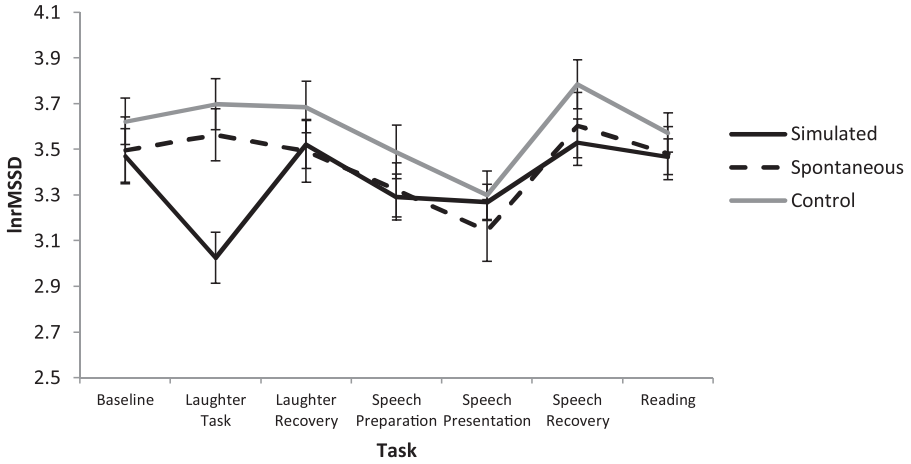

Fig. 3. Mean lnrMSSD across tasks split by condition.

was not a significant predictor ($b = -0.10$, $\beta = -.08$, $t^{63} = -0.49$, $p = .626$). Adding condition to the model explained an additional 9% of variance in lnrMSSD during the laughter task ($\Delta R^2 = .09$). This step indicates that condition explains further variance in lnrMSSD during the laughter task when controlling for known covariates and the amount of laughter produced.

4. Discussion

This study hypothesised that the cardiovascular effects of spontaneous and simulated laughter would be the same. Contrary to this hypothesis, simulated laughter resulted in a larger increase in HR and decrease in rMSSD than spontaneous laughter. It was also hypothesised that both forms of laughter would attenuate the stress response compared to a control condition. However, only the simulated laughter condition had an attenuated stress response to the TSST as represented by a smaller increase in HR than the other two conditions.

The amount of laughter produced was higher in the simulated laughter condition, and significantly predicted both HR and rMSSD. It was shown that simulated laugher increased HR more than spontaneous laughter because of the amount of laughter produced. However, the effects of simulated laughter on rMSSD were not fully explained by the differences in laughter production. During the stress task, neither the amount nor type of laughter were significant predictors of HR or rMSSD. These findings only partially support the hypothesis that the amount of laughter would predict the cardiovascular outcomes and adding condition as a predictor would not augment the predicted variance.

It appears that during laughter, the intensity and frequency of laughter increases the body's energy expenditure causing an elevated HR, akin to the effects of exercise.[26] It is known that the more exercise produced, the greater the cardiovascular changes that will occur.[27]This suggests that if the spontaneous laughter task was performed at the same frequency and intensity as the simulated laughter task, a similar increase in HR could be expected. Past research on laughter's effects on HR has only considered the effect of spontaneous laughter. For example, Averill[18] and Sugawara and colleagues[19] found that spontaneous laughter acutely increased HR. The results of this study add to previous research by suggesting that simulated laughter may have stronger effects on HR and HRV than spontaneous laughter, and the reduction in rMSSD may not be solely due to the amount of laugher produced. More research is needed to corroborate these findings.

A stress buffering effect of laughter was observed; the simulated laughter condition appeared to have an attenuated increase in HR in response to the TSST. However, this finding could not be explained by either group allocation or LIFS scores. This finding could be explained by the law of initial values which states that the extent of a physiological response is dependent on the initial level of that response[28] The simulated condition had significantly increased HR during the laughter task. As the heart was already working hard during the laughter period; the physiological stress response from the TSST may have been blunted. Past research into the stress buffering effects of laughter has only explored how laughter leads to a reduction in circulating stress hormone levels.[5,6]

There is a natural relationship between HR and HRV called cycle-length-dependence, which could partially explain the decreases in rMSSD which accompanied the increases in HR.[29] However, during the speech task, rMSSD did not decrease alongside the increase in HR, suggesting that there is an effect of laughter on HRV beyond cycle-length-dependence.

These findings do not support the Motion Creates Emotion Theory, as differences in cardiovascular effects of spontaneous and simulated laugher were observed that could not be fully explained by the amount of laugher produced. The findings, however, do support the theory that laughter acts on the cardiovascular system in a similar way to physical exercise. Laughter increased HR and decreased rMSSD, as would be expected by exercise. As the amount of laughter produced increased, so did the changes in the cardiovascular variables. This study has several limitations. The sample was young and mainly derived from University students and the results may not generalise outside this population. The laboratory setting limited the ecological validity of the task. It is also likely the participants in the simulated condition found it uncomfortable laughing in a laboratory room in front of a camera. This may have increased stress and embarrassment and therefore minimised the effects observed and may be another possible explanation for the increased HR in this group. Further research could remove the camera to reduce this possibility or measure embarrassment as a possible mediator.

More research is needed to fully elucidate the mechanisms behind the effects of laughter on cardiovascular variables. Future research could also examine whether there are any long-lasting effects on the body caused by the accumulated effects of laughter from multiple sessions over time, rather than just one short session. A replication of this study with a patient group could also extend the generalisability of the results to patient samples.

5. Conclusion

In conclusion, this study provides initial evidence that simulated laughter produces stronger cardiovascular responses than spontaneous laughter. The findings support the theory that laughter acts as a form of exercise, with more frequent and intense laughter producing a greater exercise effect on the body as indicated by increased HR and decreased rMSSD.

Conflict of interest

The authors state no conflict of interest.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial or not-for-profit sectors.

Acknowledgements

The authors would like to thank all the study participants for their participation in the study. A special thanks also to Anel Kieser and Kaula Knoche for their input with the design of the study.

Appendix A. Laughter and Intensity Frequency Scale (LIFS)

Laughter and Intensity Frequency Scale (LIFS)

Rate each subject's responses in 1 min time intervals across the 6 min period of time using the 2 scales below. Total the 6 ratings in each scale to get an estimate of both the intensity and frequency of laughter. An overall laughter score can then be determined by combining the totalled frequency and intensity scores.

Laughter Frequency Scale

0 = No laughter
1 = Rare laughter (4 or less laughs)
2 = Intermittent laughter
3 = Nearly continuous laughter

Laughter Intensity Scale

0 = No laughter
1 = Giggle/snicker (first emergence of laughter sound but still controllable)
2 = Laugh (involves facial and thoracic muscles and originates within the chest)
3 = Belly laughter or other involuntary body responses alongside laughter

Subject Number	Scale	0–1:00	1:00–2:00	2:00–3:00	3:00–4:00	4:00–5:00	5:00–6:00	Scale Total	Overall Total
	Frequency								
	Intensity								
	Frequency								
	Intensity								
	Frequency								
	Intensity								

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