Exploring the Utility of EDA and Skin Temperature as individual Physiological Correlates of Motion Sickness

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Abstract— Motion sickness (MS) is known to be a potentially limiting factor for future self-driving vehicles – specifically in regards to occupant comfort and well-being. With this as a consideration comes the desire to accurately measure, track and even predict MS state in real-time. Previous research has considered physiological measurements to measure MS state, although, this is mainly measured after an MS exposure and not throughout exposure(s) to a MS task. A unique contribution of this paper is in the real-time tracking of subjective MS alongside real-time physiological measurements of Electrodermal Activity (EDA) and skin temperature. Data was collected in both simulator-based (controlled) and on-road (naturalistic) studies. 40 participants provided a total of 61 data sets, providing 1,603 minutes of motion sickness data for analysis. This study is in agreement that these measures are related to MS but evidenced a total lack of reliability for these measures at an individual level for both simulator and on-road experimentation. It is likely that other factors, such as environment and emotional state are more impactful on these physiological measures than MS itself. At a cohort level, the applicability of physiological measures is not considered useful for measuring MS accurately or reliably in real-time. Recommendations for further research include a mixed-measures approach to capture other data types (such as subject activity) and to remove contamination of physiological measures from environmental changes.

Index Terms— Biometrics, Driver State Monitoring, Human Factors, Motion Sickness, Physiology.

I. INTRODUCTION

Despite varied research projects, there is still limited consensus about the existence of a reliable physiological measurement of motion sickness onset. Motion sickness is a complex multi-faceted condition with a fair degree of disagreement and confliction within, and between, various research projects. Motion sickness reveals itself as both a physiological and psychological condition, and the breath of human differentiation between the manifestations of these makes the field complex and often disputed. The fundamental psychophysiological explanation for motion sickness is currently best explained through the sensory conflict theory [1] which dictates mismatches between senses (visual, vestibular and somatosensory) is responsible for the onset of the condition known as motion sickness. Later work proposes the evolutionary hypothesis [2] to justify the body’s reaction to motion sickness. Specifically, it is suggested that when the body notices a mismatch in senses it assumes a poison has been ingested and it is this poison which is responsible for the mismatch. The body attempts to resolve this through getting rid of this suspected poison through sweating, burping and vomiting accompanied by thermoregulatory responses in an attempt to self-preserve. The impact of this motion sickness on subjective well-being is well understood with known symptoms such as headaches, sweating, nausea, vertigo and of course vomiting. Aside from the theoretical understanding of motion sickness symptoms, many people have had personal experiences with motion sickness aiding in at least a basic understanding of the range of subjective symptoms. In fact, looking at just passenger carsickness, and with a sample size of 4084 it was found that 46.3% reported experiencing carsickness in the past five years [3].

The area of motion sickness research which generates the greatest attention is in the mitigation of motion sickness itself. For example, exploration of design solutions for cars [4] and ships [5] amongst others provide an insight into how one might reduce motion sickness or prevent onset altogether. In every aspect of motion sickness management, comes the need for a reliable method of measuring/tracking motion sickness. In a lab-based setting, this tracking is completed subjectively where there are a variety of rating scales through which motion sickness is measured can be used. Methods such as the Motion Sickness Questionnaire SSQ [7] have seen a great deal of support and are commonly used. However, subjective scales are of course limited by the variance of subjectivity itself. Further, for many practical consumer applications for motion sickness management/tracking it is unfeasible to answer questionnaires. In many instances, such as in various recommendations made for future ‘self-driving cars’ (which carry a significant motion sickness ‘risk’) it is recommended that motion sickness should be tracked, and when onset is measured/predicted evasive actions can ensue. Such actions may include asking the occupant to focus on the horizon, or changing the vehicle route. However, for this to work there needs to be a reliable method to measure and perhaps even predict motion sickness.

It is these two motivations (efficiency and reliability in motion sickness research, and practicality for consumer applications) that fuel the search for objective and unobtrusive motion sickness measures. Given the understanding of the symptomology of motion sickness, supported by the evolutionary hypothesis, physiological measurements seems like a logical contender for exploration. However, there currently stands no published method for measuring real-time motion sickness accurately, or reliably, based on
physiological measures. This paper will review previous related research to present the current state of the art for measuring motion sickness using physiological measures. This paper will then, using both simulator-based and on-road experimentation explore the utility of physiological measures for real-time motion sickness measurement and address the reliability of physiology for determining an individuals motion sickness state.

II. RELATED WORK

It has previously been shown that changes in non-invasive cutaneously-recorded electrocardiogram (EKG) recordings correlated with participant reports of motion sickness, [8] [9] [10]. Where the EGG involves the analysis of the stomach muscles and intestines as measured by electrical signals observed through muscular contraction(s). However, the practicality of the measurement renders it unfeasible for most applications. When the EGG measurement is being taken, participants cannot move as the contractions in muscles and movement will distort the results. It is often recommended that participants lie supine when the EGG assessment is being conducted – clearly a limiting factor for many future applications, particularly automotive. Therefore, despite its apparent utility, this measure would be impractical for consumer and most experimental use.

Considering other physiological measurements, which are not limited to laboratory conditions, tracking of heart rate is very common. Heart rate (and derivations of) are easily measured using non-intrusive equipment from various body locations and in a variety of positions. Aside from the practicality of the measurement however, the lack of agreement between heart rate and motion sickness is well-documented [11]. Some research has shown a correlation between heart rate in participants who reported more motion sickness symptoms [12], whereas other research showed no reliable relationships between appearance of motion sickness symptoms and changes in heart rate [13]. The literature in this although often contradictory, tends strongly towards the conclusion that the application of heart rate measurements as a motion sickness indicator is ineffective. One of the primary difficulties with a correlation between heart rate and motion sickness is the confounding relationship between heart rate and many other variables. For instance, one paper found that apparent changes in heart-related data (specifically coefficient variance of Inter-beat intervals) “represented an increase of parasympathetic arousal during the development of motion sickness” [14]. Subsequent research [15] shows heart rate measurements represented parasympathetic arousal, specifically linked to vagal activity, during motion sickness development. Looking even more simplistically, it is known how heart rate is significantly affected by emotional state where even a relatively mundane computer related task can induce emotional responses which affect heart rate [16] when sitting motionless in a chair. Conclusively, heart rate and/or derived measurements alone are not thought to be useful to reliably measure motion sickness. This is due to the lack of proven direct correlation to motion sickness, and the evidence that heart rate is affected by many other factors which will make any correlation to one specific condition very difficult.

Another paper considered a breath of physiological results, and looked to see if physiological measures during an exposure, were a reliable predictor for post-exposure motion sickness state [17]. With significant post-hoc data ‘filtering’, and at a group level they report stomach activity, blinking behaviour, and breathing are useful indicators of the prevalence of cybersickness. There appears to be a correlate here, but the reliance on significant post-hoc data filtering and a lack of real-time data make it difficult to draw conclusions about the differentiation from motion sickness symptoms to symptoms of discomfort or other arousal states.

Other research shows more promise and explores the measurement of Electrodermal Activity (EDA) as an indicator of motion sickness. This seems to be a logical research area where it is known that increased sweat rate is a common symptom of motion sickness as part of the symptomology explained through the evolutionary hypothesis [2]. An early study in this area discusses the link between skin conductance due to volar sweating and susceptibility to motion sickness [18]. It was found that those who had a naturally higher sweat rate were also more prone to motion sickness. This was assessed during a sea-sickness trial where participants’ sweat rate was recorded prior to exposure and then when on the boat participants were visually assessed and questioned every half an hour for subjective motion sickness. This, although not a real-time assessment, does identify this link between physiology and motion sickness. Another study in the field of aviation showed, with a sample size of 170 participants, that increases in skin conductance correlated with subjective motion sickness – as measured with a motion sickness questionnaire after the motion sickness-inducing stimulus [19]. The authors discuss a lack of correlation between skin conductance and specific single indices of motion sickness however, admitting there are extraneous variables which are not currently understood. It is difficult to understand the relevance of this finding where motion sickness was not measured throughout exposure, rather, at the end of the exposure. In another study, participants followed prescribed head movement procedures whilst sitting in an enclosed box which was spun around on a turn-table. Participants sat in the box until they reached a common state of motion sickness and then their physiological state was assessed. This study [20] concluded how phasic skin conductance (measured from the forehead) was useful as a motion sickness indicator at a group level. Further research by the same author reported a correlation between reduction of nausea and reduction in skin conductance when researching the effectiveness of anti-motion sickness drugs [21] – again linking the physiological measure to subjective sickness. However, this later study does highlight how the skin conductance observations were likely affected by the drugs, which they were controlling – something many citations of this paper often overlook.

Conclusively, at a group level, electrodermal activity (EDA) is been shown to be related to motion sickness, and therefore is a good candidate for further exploration for real-time utility. As yet, no published paper has considered EDA as a real-time measurement, where the aforementioned literature has taken readings after exposure to a sickness-inducing experience and other have measured sweat rate in relation to a propensity to become motion sick (i.e., susceptibility based on sweat rate).

Further to EDA, temperature presents itself as another logical area for exploration, again due to the understanding of
the thermoregulatory response to motion sickness. In fact, in severe seasickness people have even been known to develop hypothermia. In less severe instances, the underlying effects still remain however. As part of the evolutionary theory or ‘toxic’ hypothesis, the body will cool core temperatures to reduce the chance of overheating, draw blood away from peripheral limbs to ensure effective circulation of core organs and promote cutaneous vasodilation to further cool the body through convection [22]. The relationship between motion sickness and thermoregulation has previously been comprehensively detailed [23]. One review paper, looking specifically at this thermoregulatory response to motion sickness does not present any original supporting data, but does conclude that measuring temperature to infer motion sickness is a worthwhile research pursuit [24]. Skin temperature is of particular interest in combination with EDA where under normal circumstances, the measures are strongly related due to the nature of human thermoregulation.

There is an intrinsic link between the physiological responses of sweating, thermoregulation and motion sickness and various sources provide evidence to support this. However, despite not presenting any data, previous work casts doubt on the utility of such measures for motion sickness predictors [25]. This previous work draws on the understanding that measures such as skin temperature and EDA (amongst others) are also affected by other emotional and environmental stimuli. This reveals a challenge in finding correlations linked to just one variable and highlights the impact that procedure, experimental events, emotional states and environments may have on these measures. With this in mind it is considered that the use of a highly controlled environment (such as a driving simulator) as well as a more naturalistic environment may afford a comparison between low emotional/environmental variability and high emotional/environmental variability.

One of the most beneficial uses for a reliable physiological measurement of motion sickness would be in the real-time tracking and perhaps even prediction of motion sickness state/onset. Uses for such a tool have previously been referenced in relation to self-driving and autonomous vehicles [26]. As the literature supports, the most common method for measuring real-time motion sickness is to ask for a subjective rating on some form of motion sickness scale, for example the FMS scale [27]. Such subjective scoring is used in most, if not all, motion sickness studies and should be considered our ‘baseline’ technique to compare other methods against. Importantly, the motivation for new motion sickness measurements comes from its potential utility in a consumer application, hence the desire to move away from subjective self-reporting. Considering this, it is important to consider the feasibility and practicality of physiological measurements from a consumer point of view. For example, considering EDA, it is unlikely people will want to wear head mounted devices, or wear devices that require the application of electrolyte gel (as has been used in some of the cited literature). Clearly, if the goal is to look for a useful ‘real-world applicable’ method of motion sickness tracking using physiology the method should be non-invasive and easy to measure for a consumer. Furthermore, the method needs to be suitable as a real-time measurement and cannot rely on post-hoc analysis and post-exposure analysis.

The primary research question has therefore been highlighted: Is it possible to correlate motion sickness in real-time to electrodermal activity and/or skin temperature using non-invasive methods at an individual or group level?

III. Method

To answer the research question, this project was spread over three user trials to both increase the quantity of data and provide two motion sickness inducing environments. Firstly, a simulator based study was conducted where physiology data could be collected in an ‘ideal’ environment. In the simulator there were no external stressors such as other road users and no change in environment, such as ambient temperature, directional temperature or humidity (all factors that may affect EDA and skin temperature). All participants experienced an identical, highly controlled scenario ensuring emotional state was as comparable as possible between participants. Secondly, a repeated-measures on-road user trial was used where participants were driven round UK roads whilst sitting in the rear of a vehicle to simulate an autonomous car experience. The on-road trial was run twice, spaced 14 days apart as required for data collection for a separate study. Although the route, duration of drive and driving style were kept the same for each participant (as well as time of day for repeat measure participants) this provide a more naturalistic environment. Having simulator and real-world allow for a comparison of ‘ideal’ vs ‘real world’ results where in the real-world there are inevitably going to be changes in directional temperature, humidity and external stressors such as other road users.

Fourteen participants were recruited for the driving simulator study which used the 3x3D simulator at the University of Warwick [28]. This is simulator uses a Range Rover evoke as the fixed-base ‘ego’ vehicle, which is situated within a 360-degree screen and is a fully immersive driver-in-the-loop simulator. Participants completed a manual driving scenario in the 3x3D, which took up to 33 minutes and included a mixture of urban, rural and motorway roads. The route was designed to be challenging considering motion sickness where it is expected that most participants will experience at least some minor symptoms of motion sickness in a fixed-base simulator. To ensure participant well-being, the route included a 5-minute familiarisation period with straight roads and slow speeds, where the gentle bends and increased speeds were introduced as the drive progressed. The final 10-minutes of the route were particularly challenging with complex bends and roundabouts designed to challenge those with low susceptibility to motion sickness. Subjective motion sickness was measured using the Fast Motion Sickness Scale (FMS) [27] which involved asking participants once per minute to rate their motion sickness on a scale of 1-20, where 20 was the most severe motion sickness. The Simulation Sickness Questionnaire SSQ [7] was also used before and after the simulator exposure. The only task for the driver during this simulator study was to manually drive the simulated vehicle safely and efficiently around the simulated world.

For the two on-road user trials a further 26 participants (completely independent from the simulator trial) were recruited, where 21 of these 26 completed two exposures as part of the repeated-measures study. Participants were driven
round a pre-determined route for up to 31 minutes by a trained driver trying to maintain a comparable driving style between each drive. This route involved a mixture of urban, motorway and country roads. The vehicle used was a right-hand drive, 2018 Land Rover Range Rover Sport L494 with no window tint. This trial required participants to be driven on the same route twice in total; with each exposure exactly 14-days apart and at the same time of day. Five participants withdrew after the first drive, leaving only one data set for these participants. During the drives, participants sat in the rear near-side passenger seat and completed a reading task on the head-rest mounted screen installed in the Range Rover. This reading task is to ensure that participants are all behaving similarly with regards to eye glance fixation [1] as well as to control for well-known motion sickness mitigation strategies (such as looking at the horizon to avoid sensory conflict). The NASA Task Load Index (TLX) [29] was used to validate that the reading tasks did not have an impact on workload (where participants were given two reading tasks in a random order between the two exposures).

The Empatica E4 wristband [30] was used to collect physiological data from the participants throughout both user trials. The E4 is a wireless wrist-worn device requiring no electrolyte gel. This device was used as it is a non-invasive method and was considered appropriate considering the focus for a consumer-practical method, while providing accurate and reliable physiological data which is FDA approved [27]. The data was processed using the method described in [31]. The Empatica E4 measured EDA in the unit of in micro-Siemens (µS) and skin temperature in the unit of degrees Celsius (°C), both at a rate of 4Hz. Each participant provided a subjective motion sickness score every 60 seconds as per the FMS. Each participant had at least two minutes resting time before starting either the on-road or simulator driving scenario where they sat calmly in the car seat - this data was used to infer a physiological baseline for each participant. The ambient temperature within the simulator and on-and vehicle was kept constant throughout all exposures at 21 degrees Celsius, with windows remaining closed for the entire on-road study.

The data for all participants was processed by calculating mean skin temperature and EDA score for the minute leading up to the request for FMS (once per minute subjective MS score). For example, the driver started at time 0 and the FMS score was given at minute 1. The comparative physiological figure to compare against minute 1 FMS score was taken as an average over the entire driving scenario of 32.298°C, SD=1.531. Therefore, Spearman’s correlations will be used for the majority of data analysis. Such a method can be calculated minute-by-minute if required and does not require any post-hoc manipulation of the data. Other methods such as measuring phasic response require identifying peaks and troughs, which are only identifiable upon the presentation of a completed data set and thus are not useful for real-time applications.

### IV. RESULTS

Three groups have been established within this data set for analysis.

- **Group 1:** Simulator study participants (N=14)
- **Group 2:** On-road study participants drive 1 (N=26)
- **Group 3:** On-road study participants drive 2 (N=21)

Group 1 contained 14 participants including seven females and seven males with mean age of 30 (SD=10.69). Of the 14 participants recruited, seven dropped out of the study mid-way due to motion sickness (six females and one male). The average drive time was 21 minutes where the shortest drive was 8 minutes (due to dropping out) the longest drive was 33 minutes. The physiological data of participants who dropped out was retained for the analysis, but trimmed to the point time in which they ended the driving scenario. This user trial was not concerned with recovery, but rather motion sickness onset therefore it was unethical to continue to collect subjective data once the participant had asked to end the study so data collection ended if the participant asked to end the study.

Group 2 contained 26 participants including 14 females and 12 males with a mean age of 33.6 (SD=12.8). The average drive time was 28 minutes where the shortest drive was 27 minutes and the longest drive was 31 minutes and there were no dropouts.

Group 3 contained 21 participants with a mean age of 31.1 (SD=11.8), the average drive time for Group 3 was 28 minutes where the shortest drive was 27 minutes and the longest drive was 30 minutes. The change in drive times for Group 2 and 3 was due to slight changes in road traffic, which were not measured or controlled other than by time taken to complete the route.

For Group 1 the average EDA score during the resting period (i.e. baseline) was 0.782 µS, SD=1.063, where during the driving scenario this increased to 1.015 µS, SD=1.531. The average skin temperature score during the 2-minute resting period was 32.644°C, SD=0.935, which decreased to an average over the entire driving scenario of 32.298°C, SD=1.528.

For Group 2 the average EDA score during the resting period was 0.669 µS, SD=0.635, where during the driving scenario this increased to 1.092 µS, SD=1.683. The average skin temperature score during the resting period was

The method of data analysis must be considerate of the motivations of this study – that is to explore the utility of physiological measures in ‘real-time’. For any practical method one must be able to measure motion sickness in a similar epoch as the subjective measure of one minute. Therefore, Spearman’s correlations will be used for the majority of data analysis. Such a method can be calculated minute-by-minute if required and does not require any post-hoc manipulation of the data. Other methods such as measuring phasic response require identifying peaks and troughs, which are only identifiable upon the presentation of a completed data set and thus are not useful for real-time applications.

\[
\Delta \text{EDA}_{\text{minute } x} = \text{EDA}_{\text{minute } x} - \text{EDA}_{\text{baseline}} \tag{1}
\]

\[
\Delta \text{Skin Temperature}_{\text{minute } x} = \text{TEMP}_{\text{minute } x} - \text{TEMP}_{\text{baseline}} \tag{2}
\]
For Group 3 the average EDA score during the resting period was 0.685 μS, SD=0.262, where during the driving scenario this increased to 1.225 μS, SD=0.043. Similarly, the average skin temperature score during the resting period was 32.368°C, SD=1.448, which decreased marginally to 32.061°C, SD=0.431, whilst driving.

As mentioned, participants in Groups 2 and 3 each completed a basic reading task, to ensure consistency in behaviour. The order in which participants received the reading task was randomised between participants and exposures. To ensure this task did not impact results a paired T-Test was used to understand if either of the tasks required more workload. The T-Test revealed there was no significant difference between RTX scores for the two reading tasks t(21)=1.123, p=0.283 so the analysis continued.

The figures below show combined data for both EDA and skin temperature (mean minute-by-minute measure for the whole group) plotted against mean subjective MS rating (FMS). Figure 1 shows data for Group 1 (simulator trials), and Figure 2 combines Groups 2 and 3 (on-road trials).

Looking at the graphs presented in Figures 1 and 2 there are some visual similarities between the relationship between the physiological measures and the subjective scoring. The error bars are calculated from standard error reveal variability within the group, which appears to increase as time progresses. To explore the relationship between these measures at a group level, average FMS score and average physiological score for the entire group was calculated and explored for correlation and presented in Table 2. The table has been colour coded to highlight the correlations which were significant with 99% confidence in green, 95% in orange and no significant correlation in red.

Table 2 identifies significant and strong correlations for four out of the six correlations across the groups. This data shows the average FMS score for the group per minute compared against the average delta EDA and skin temperature measure. The direction of the correlations are as expected for EDA (all groups) as well as TEMP for Group 1 and Group 2. However, the significant positive correlation for Group 3 TEMP is in contrast to the expected negative correlation a seen in Groups 1 and 2.

Group level data is not of great interest for this research. Therefore, the next step was to explore the relationships between physiology and subjective ratings at an individual level. The data was analysed to see if EDA and/or skin temperature were correlated to the subjective FMS score by looking how each measure changes in relation to the other for each individual participant. The Shapiro Wilk test was first used which showed none of the three group’s data sets’ were normally distributed where p<0.05 in all instances. Therefore, for both Δ EDA and Δ skin temperature a Spearman’s rank-order correlation was run to determine the relationship of the physiological measures and the subjective motion sickness score (FMS). The results from this correlation analysis have been presented overleaf in Table 2. The data presented in Table 2 lists all the correlations (spearman’s $r_s$) of each participant’s physiology (‘EDA’ and ‘TEMP’) against their FMS score, where ** denotes a 99% confidence rating and * denotes a 95% confidence rating. The table has been colour coded to highlight the correlations which were significant with 99% confidence in green, 95% in orange and no significant correlation in red.

To summarise the number of significant and non-significant correlations observed in Table 2, Table 3 has been created. Table 3 presents the total numbers of each correlation (or lack thereof) for each group and presents the percentage of each along with a total which combines Groups 1, 2 and 3.
Correlations of EDA / Skin Temperature (TEMP) against FMS

<table>
<thead>
<tr>
<th>Group</th>
<th>Participant</th>
<th>r, FMS - EDA</th>
<th>r, FMS - TEMP</th>
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<tbody>
<tr>
<td>175</td>
<td></td>
<td>.815**</td>
<td>.166</td>
</tr>
<tr>
<td>180</td>
<td></td>
<td>-.865**</td>
<td>-.814**</td>
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<tr>
<td>195</td>
<td></td>
<td>.066</td>
<td>.238</td>
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<tr>
<td>237</td>
<td></td>
<td>.847**</td>
<td>-.941**</td>
</tr>
<tr>
<td>388</td>
<td></td>
<td>.145</td>
<td>.084</td>
</tr>
<tr>
<td>489</td>
<td></td>
<td>.232</td>
<td>.894**</td>
</tr>
<tr>
<td>549</td>
<td></td>
<td>-.647**</td>
<td>-.603**</td>
</tr>
<tr>
<td>607</td>
<td></td>
<td>.181</td>
<td>-.651**</td>
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<td>633</td>
<td></td>
<td>-.365</td>
<td>-.079</td>
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<td>731</td>
<td></td>
<td>-.334</td>
<td>-.115</td>
</tr>
<tr>
<td>784</td>
<td></td>
<td>-.228</td>
<td>-.825**</td>
</tr>
<tr>
<td>846</td>
<td></td>
<td>.627**</td>
<td>-.684**</td>
</tr>
<tr>
<td>950</td>
<td></td>
<td>.982**</td>
<td>.746</td>
</tr>
<tr>
<td>968</td>
<td></td>
<td>.353</td>
<td>.263</td>
</tr>
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</table>

**=99% confidence rating, *=95% confidence rating

<table>
<thead>
<tr>
<th>Group 1</th>
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</table>

Table 3 above shows no coherent trend towards a collective of significance with high proportions of the measures showing insignificance (41% for EDA and 39% for TEMP). Looking at just the significant results, correlations ranged from +0.982 to -0.865 for EDA and from +0.894 to -0.941 for skin temperature. This is a very large range where correlations are ranging from almost a perfect positive (+ve) correlation to an almost perfect negative (-ve) correlation between participants for the same measure. To present this range of correlations graphically, Figures 3 and 4 below plot these correlations on histograms:

Fig. 3. Correlation values for EDA vs FMS

Fig. 4. Correlation values for skin temperature vs FMS

The range of correlation values is a strong indicator that there is no trend here. However, in order to better quantify
the overall correlation of this data at an individual level, averages correlations were calculated. It is not possible to simply take the mean from all correlation figures, so instead methods for averaging correlations were explored. One common method for achieving this is by transforming the data into a Fisher’s z score, calculating the average of that, and then transforming the data back. The calculation of Fisher’s Z is a common method of transforming the data into an approximate normal distribution. Another method involves averaging observed sample r, correlations [33] – which has been concluded to be a superior method [34] and is presented below:

$$\bar{r} = \frac{\sum(n_i - 1) \left( r_i + \frac{r_i(1 - r_i^2)}{2(n - 3)} \right)}{\sum n_i - k}$$

(3)

The above formula, is considered to be an effective method of taking the average correlation from a group (where k is the number of individual samples) and is perfectly suited to this task. As a slight critique of the notation of this equation in its current structure, the equation within the parenthesis on the right needs to be calculated before being multiplied by the constant $\frac{\sum(n_i - 1)}{\sum n_i - k}$. By nature however, this interpretation produces a new correlation value for each participant, to which a standard averaging calculation would then have to be applied. It is clear from the derivation of this equation, as presented by the original author [35], that $\bar{r}$ should be one single value denoting the average of all correlations. Thus it is logically easy to misinterpret the use of the equation proposed above. Through understanding the derivation of this from original equations, it became clear that the equation could be presented as follows as a more ‘up to date’ notation:

$$\bar{r} = \frac{\sum(n_i - 1) \left( r_i + \frac{r_i(1 - r_i^2)}{2(n - 3)} \right)}{\sum n_i - k}$$

(4)

Using the revised equation above (adapted from [34]) the average correlation figure for each group and physiological measure (as well as for all participants combined), has been calculated and presented below:

<table>
<thead>
<tr>
<th>Table IV</th>
<th>AVERAGE CORRELATIONS FOR GROUPS 1, 2 AND 3</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Average Correlation Coefficient ($r^*$)</td>
</tr>
<tr>
<td></td>
<td>EDA vs. FMS</td>
</tr>
<tr>
<td>Group 1</td>
<td>-0.03</td>
</tr>
<tr>
<td>Group 2</td>
<td>0.11</td>
</tr>
<tr>
<td>Group 3</td>
<td>0.20</td>
</tr>
<tr>
<td>Combined</td>
<td>0.12</td>
</tr>
</tbody>
</table>

Looking at the average correlations ($\bar{r}^*$) for each group, the strongest correlation given is -0.3 for Group 1 skin temperature vs FMS which is considered to be a very weak correlation. Overall, none of the correlations were found to be of any notable strength and are also considered to be very weak – unsurprising considering the variance observable in Table 2. When combining all groups, the average correlation across the entire sample size for EDA is found to be 0.12, and for skin temperature it is 0.15.

Despite the above method being considered to be the most unbiased and useful method for averaging correlations [34], the method of using Fisher’s z score is still more commonly used (perhaps due to convenience). There is little interest for this work to compare the benefits of either methods, but to ensure comprehensive analysis of results, the Fisher’s Z method was also completed. This involves transforming each rs into a z score, by using the following formula:

$$z_i = \tanh^{-1}(r_i)$$

(5)

Then, averaging these z values as follows:

$$\bar{z} = \frac{\sum(n_i - 3)z_i}{\sum(n_i - 3k)}$$

(6)

And finally, transforming $\bar{z}$ to $\bar{r}$ by:

$$\bar{r}' = \tanh(\bar{z})$$

(7)

An explanation as to the utility of this method is presented [34], along with further details of the notation where k is the number of individual sample rs being aggregated. There is still support of this method within more modern research, where recent papers are still using this technique for similar tasks [36]. The results from this method have been presented below in Table 5:

<table>
<thead>
<tr>
<th>Table V</th>
<th>AVERAGE CORRELATIONS FOR GROUPS 1, 2 AND 3</th>
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<tbody>
<tr>
<td></td>
<td>Average Correlation Coefficient ($r^*$)</td>
</tr>
<tr>
<td></td>
<td>EDA vs. FMS</td>
</tr>
<tr>
<td>Group 1</td>
<td>-0.04</td>
</tr>
<tr>
<td>Group 2</td>
<td>0.06</td>
</tr>
<tr>
<td>Group 3</td>
<td>0.18</td>
</tr>
<tr>
<td>Combined</td>
<td>0.02</td>
</tr>
</tbody>
</table>

The results presented in Table 5 are quite similar to that which is presented in Table 4, showing there is some difference between these two methods, but the conclusions taken are similar with no signs of any useful correlations.

Finally, as a note to further validate the method of subjective motion sickness used in this paper, the FMS data was compared against the SSQ for the simulator study, and the MSAQ for the on-road study. As per the FMS author’s recommendations [27], the FMS peak score (i.e., the maximum score given by each user) was used for this overall comparison of the subjective measures. Using a Spearman’s rank-order correlation it was shown the SSQ and FMS peak were strongly and significantly correlated with a sample size of 14 ($r_s=0.742, p<0.001$) for the simulator participants. For the on-road study the MSAQ and FMS peak were also shown to be significantly and strongly correlated with the combined sample size of 43 ($r_s=0.838, p<0.001$).

V. DISCUSSION

This paper looked to build upon the previous literature e.g., [18] which suggests there is a link, and therefore a useful
correlation between physiological measures and motion sickness. This concept is theoretically supported by the evolutionary hypothesis [2] which explains how increased sweat rate and temperature change are the effects of evolutionary-developed coping mechanisms for self-preservation when in a state of sickness. In general, when suffering from motion sickness one would expect to see an increased sweat rate and a decreasing skin temperature. Looking initially to the graphs depicted in Figures 1 and 2 there does indeed appear to be some visual relationship, where the average scores of each group are presented. Indeed some significant correlations are found at group level and have been presented in Table 2. Out of the six correlations performed (between three groups and two measures), two correlations were found to be insignificant Further, looking at the direction of correlations for Group 3 FMS vs. Skin temperature a significant positive correlation was found. This is in opposition from what one might expect to see, through the understanding of the thermoregulatory response to motion sickness and self-preservation [23]. Despite this correlation being significant, one should interrogate this as a spurious correlation and disregard this as evidence for the relationship between the measure of motion sickness and skin temperature. Previous research has identified the relationship between similar physiological measures and post-exposure motion sickness measurement [17] [18] [20] [24] and the data presented in this paper presents, for the first time, this comparison using a real-time analysis. The findings in this paper are not entirely in support of a clear-cut correlation and with mixed results it is understandable why the literature in this area is so mixed also. It would seem from the correlation values that EDA has a slight edge on skin temperature for group-level analysis – yet it would be wrong to conclude that either of these measures, at this stage, are considered reliable.

The challenge with group-level analysis for physiological measures is the variance within-subjects is lost when averaging the scores across a group. The error bars within Figures 1 and 2 give an indication of this increasing variability as time progresses – and this is vitally important for truly understanding the utility of these measures. It was important therefore to consider how these measures correlate on an individual basis – exploring therefore the true reliability of these measures for a real-time predictor of motion sickness. Individual Spearman correlations were run for each individual, for both EDA and Temp. The list of correlations has presented in Table 2 and highlights immediately a great variation in both significance and direction of correlations. Looking at all the participants across all the groups (62 data sets across 42 participants) only 54% of correlations were shown to be significant (see Table 3), and of those, the correlation coefficients for EDA ranged from +0.982**(Group 1 participant 950) to -0.890**(Group 2 participant 217). Similarly, for skin temperature correlations ranged from +0.939**(Group 2 participant 217) to -0.941**(Group 1 participant 237). There is a considerable range in observed, and statistically significant correlations between participants. This range of correlations is evidenced by the scale of the error bars observed in the graphs (Figures 1 and 2) and have been plotted using histograms in Figures 3 and 4 to show the range of correlations from (+ve) to (-ve).

The observations at an individual level further question the utility of this physiological data as a predictor of motion sickness for an individual. It is the variance between participants which is perhaps the most useful indication that these physiological measures are not an accurate predictor of motion sickness state.

Despite the evidence already presented, it was still interesting to look for the average correlation for each group and the entire sample. These averages have been calculated using a revised equation as presented in [34]. The results from this study of both simulator and real-world driving (presented in Table 4) show that, on average, the correlations for each category are very weak, with the greatest correlation being -0.3 for the relationship between subjective motion sickness and skin temperature. Although the aforementioned variance is perhaps the most telling metric here, these correlation averages are insightful for forming a quantitative conclusion. These correlations indicate that physiological measures for skin temperature and EDA are not useful as a predictor of real-time motion sickness across a population, agreeing, with and providing evidence to support the aforementioned literature [25].

One reason why this data set (comprising of 62 data sets from 40 participants) showed no useful correlation between subjective motion sickness and physiological signs, was considered be due to individual differences between participants. There were, after all, 66 significant correlations found (54%) between the three groups and two physiological measures. It was theorised initially that perhaps some people are more suited to being measured for physiology than others, where for some participants their EDA/skin temperature may be a reliable measure, but for others it is less so. This theory was based on understandings such as physical fitness in an effector for both motion sickness susceptibility [68], and sweat rate / thermoregulation [69], so it is perhaps individual characteristics affect an individual’s measurable physiological response. To explore this, it is possible to compare Group 2 participants to their second set of scores in Group 3 using Table 2. Where (besides dropouts) Group 2 contained the same participants as Group 3 with the repeat drives taking place 14-days apart. In Group 2, participant 146 provided non-significant correlations of 0.092 for EDA and -0.276 for skin temperature, but on their second drive in Group 3 their respective correlations were 0.751** and 0.633** both significant with a confidence level of 99% (as denoted by **). This miss-match in correlations within participants is seen throughout the data set presented in Table 2. Out of 21 participants, there is only one participant who showed significance for both physiological methods for both exposures (participant 217). However, despite significance, the correlation for EDA changed from -ve to +ve across exposures, and skin temperature correlation changed from +ve to -ve across exposures. This means that although significant correlations, they are clearly not reliable as a motion sickness predictor as they are directly opposed to each
other considering direction. This change in direction of correlation is seen within many of the participants presented in Table 2. Given these disagreements between repeated measures, there is no evidence in this data set to support the idea that physiology is reliable across exposures for the same individual – although it must acknowledged that participants only experienced two exposures.

One common challenge with using physiological measures such as EDA and skin temperature is the propensity for these measures to be impacted by external factors. It is known that various states of arousal can induce a physiological change [19] where the term ‘arousal’ covers various emotional states. One key text specifically looking at physiological correlates of motion sickness summarises how most researchers agree that the physiological responses of motion share many of the component characteristic of stress or alarm [25] (p.164). The cited author explores this idea further to explain how the stress response specifically impacts both EDA and skin temperature. Further key texts also reveal correlations between the stress response and EDA [37]. Considering a hypothetical use case it is easy to imagine how mood, excitement, stress, fatigue – as well as countless other emotions or arousal states experienced both in everyday life, as well as driving, will affect these physiological measures.

Further to emotional states, these measures are also not independent of the environment, where environmental conditions are also able to affect these physiological readings. Aspects such as directional temperature, ambient temperature, humidity, airflow and clothing for example all will affect EDA and skin temperature. Actions such as turning on air conditioning, opening a car window, driving from shade to sunlight etc. will all have a considerable impact on EDA and skin temperature – further complicating any direct relationship between these measures and motion sickness for a consumer application.

Following this understanding, it is apparent why it is challenging to correlate just motion sickness as an isolated measure from these physiological measures which are by their nature affected by a range of factors. This was understood before this research took place, yet it was unknown (and unquantified) exactly how much other factors would impact this data. To somewhat cater for this unknown scale of impact from external variables, this user trial benefited from two motion sickness inducing environments – a driving simulator and an on-road experiment. The simulator experiment was entirely controlled with identical environmental conditions and identical emotional stimulus between participants (e.g., the simulated world was identical for each participant and both exposures). Previous research had proposed that these measures are impacted by environmental and emotional changes [25] and the data presented in this current paper is able to provide some quantitative insights into the predicted conclusions from [25]. It is perhaps reasonable to hypothesise that the simulator participants (Group 1) would show stronger correlations with greater quantities of significance, compared to the more naturalistic environments from Group 2 and Group 3. However, this data research sees no evidence for this. In fact, Group 1 participants in the simulator provided the greatest number of insignificant correlations for both EDA and skin temperature – compared to Groups 2 and 3. Showing that even in a controlled environment, these measures are greatly impacted by external valuables and thus are unreliable to use as motion sickness measures. Comparing this to the existing literature, it has already been discussed that even very mundane computer tasks evoke physiological responses [16] and other research has shown how physiological measures such as breathing, blink rate and EGG all had a significant interaction affect with display type (comparing head mounted to traditional displays) [17].

There is no argument that motion sickness is related to the physiological response of sweating and thermoregulation. However, the measurement of these seems to be impacted more so by environment and emotional factors than it does motion sickness. Not only are the measures discussed affected by emotional states, but they are also heavily interconnected through the process of thermoregulation. When hot, the human body will increase sweat rate (as measured through EDA) to help cool the skin through evaporative heat loss – so these factors are certainly not independent of one-another.

The research presented in this paper evidences the importance of appropriate sample sizes for physiological studies, where some participants had a very strong and significant correlation between their physiological state and motion sickness. Therefore if looking at just a few of these participants one may falsely misinterpret the true relationship when generalising across a population.

VI. CONCLUSION

This research measured real-time subjective motion sickness alongside electrodermal activity (EDA) and skin temperature across simulator-based and on-road user trials. The trials contained 14 participants using a vehicle simulator, and 26 participants taking part in an on-road motion sickness study, where 21 of those participants completed the on-road study on two separate occasions separated by 14 days. In total, 294 minutes of vehicle simulator data was collected along with 1316 minutes of on road data, providing a total sample of 1610 minutes across 40 individual participants and 61 separate data sets. The physiological measures of EDA and skin temperature were compared to participants’ self-reported motion sickness state using the FMS scale - which has been validated within this trial, and within previous literature to be significantly and strongly correlated to motion sickness state. The group level analysis revealed some relationships between the physiological measures of EDA and skin temperature with motion sickness – although certainly not conclusive for a group level. At an individual level a very mixed set of results were found where only just over half (54%) of correlations were significant, and of those, there was a wide range of correlation strengths and directions for both physiological measures. Correlation results presented in this paper evidence that there is no coherent or
reliable relationship between these physiological measures and subjective motion sickness at an individual level. Although, this range in correlation strengths and direction is perhaps the most useful indicator of the utility of these measures, the average correlation for each measure is further proof that these measures are not useful for motion sickness prediction/measurement. On average, it was shown that FMS correlated to EDA at $r_s=0.12$ and with skin temperature at $r_s=0.15$ – both are considered incredibly weak.

This study is the first to present real-time subjective motion sickness ratings correlated with real-time physiological data. Although there is no dispute of the fundamental interconnected nature of motion sickness and EDA/skin temperature - this study can conclude that these measures are entirely unreliable for the measurement and/or prediction of subjective motion sickness state. Possible reasons for this lack of correlation have been discussed within the paper, notably the impact of external factors such as stress emotional states and environmental conditions have on these physiological measures. Comparisons were made between the highly controlled simulator environment and the naturalistic on-road study which evidenced even in a highly controlled simulator, there was no evidence of reliability for these measures as a motion sickness predictor.

This paper closes the debate on the utility and reliability of physiology as a predictor of motion sickness by analysing the relationship in real-time. It acknowledges that, at a group level for some participants EDA and skin temperature may be related to motion sickness, but evidences that they are not reliable enough infer motion sickness state. Individual analysis is even less reliable and this paper shows that any recommendations for correlations at group-level are not appropriate to draw conclusion on at an individual level. Future research may consider the scope for filtering physiological data to control for other variables such as alarm, stress, environment and thermoregulation. However, given the breath of effectors it is likely to be a significant challenge. Anecdotally, the researchers of this user trial, and other simulator sickness studies have noticed visual cues which help them understand when someone is becoming motion sick during experimentation. Actions such as wiping their forehead, putting their hand to their mouth and deep and prolonged exhales were observed in many participants who soon after ended a study due to severe motion sickness. A mixed methods approach using camera-based detection of such actions could be considered for further research to objectively identify motion sickness onset. Combining multiple data sources including activity, actions, predisposition to sickness and physiological state among others may provide a method to measure motion sickness objectively. However, for now, it is concluded that despite the intrinsic relationship, physiological responses are an unreliable and entirely ineffective method of measuring/predicting real-time motion sickness for individuals.

VII. REFERENCES

[13] A. Graybiel and J. R. Lackner, "Evaluation of the Relationship Between Motion Sickness Symptomatology, and Blood Pressure, Heart Rate,


[71] B. S. Cheung, K. E. Money and I. Jacobs, "Motion Sickness Susceptibility and Aerobic Fitness: A Longitudinal Study," Aviation, Space, and Environmental Medicine, March 1990.