Molly L McClelland, Channing S Sesoko, Douglas A MacDonald, Louis M Davis

University of Detroit Mercy, Detroit, United States

Effects on vital signs after twenty minutes of vaping compared to people exposed to second-hand vapor

Abstract

Introduction: Very little is known about the immediate physiological implications of vaping or inhaling second-hand vapor. This study used a quantitative approach to understand the short-term physiological implications of vape use and exposure to second-hand vapor for people who do not vape.

Material and methods: One hundred and forty-eight people participated in the study, 75 self-identified as non-vapers and 73 self-identified as people who vape. All participants were over the age of 18. Participants used or were exposed to non-flavored e-juice without nicotine in Sorin® vape devices. Heart rate, blood pressure, respiratory rate, blood oxygenation, blood glucose and pulmonary function tests were assessed. Physiological parameters were assessed prior to vape use or exposure to vapor and again after 20 minutes of vaping.

Results: Findings indicated there were no significant changes in most health parameters except blood pressure which was reduced in both groups. Heart rate was also significantly reduced for vaping participants.

Conclusion: Vaping without flavorings or nicotine do not appear to have an immediate negative health impact on vital signs. The physiological effects of long-term exposure and/or vape use requires additional investigation. Information was established regarding the physiological effects of non-flavored, non-nicotine vaping so future studies can compare the effects of vaping with assorted flavors and nicotine concentrations to the effects of vaping only the base ingredients (vegetable glycerin and propylene glycol). New knowledge was gleaned relating to exposure to vapor, a phenomenon not previously examined but common especially among non-vaping people who attend social events where people are vaping.

Key words: vaping, second-hand vapor, e-cigarettes, e-juice, vital signs

Adv Respir Med. 2020; 88: 504–514

Introduction

The public health impact of vaping has become a frequently discussed topic [1] but very little is currently known about the immediate physiological impact of vaping or second-hand exposure to vapor. The paucity of information related to the physiological effects of vaping is presenting problems for the medical community in knowing how to advise patients regarding vape use. This study was aimed at investigating the immediate physiological effects of vape use compared to second-hand exposure to vapor and to also better understand the demographics between the two groups.

Patented in 2003 but developed in 1963, vaping was established as an alternative to cigarette smoking [2]. The incidence of vape use is on the rise [3, 4]. and has recently been subjected to regulations under the United States (US) Federal Food, Drug and Cosmetic Act [5]. The new regulations include restricting sales to minors and listing health warnings on labels. US Federal regulations were implemented because, despite not containing any tobacco, the US Center for Disease Control and the US Federal Drug Administration consider vapes to be tobacco products and potentially harmful to health [3, 6, 7].

Since vaping is a fairly new phenomenon, there remains a lack of research regarding the

Address for correspondence: Molly L McClelland, University of Detroit Mercy, Detroit, United States; e-mail: mcclelml@udmercy.edu

DOI: 10.5603/ARM.a2020.0148
Received: 18.05.2020

ISSN 2451–4934

effects of its use and medical implications. Some reports suggest that vaping helps people quit smoking [9, 10]. Other reports suggest that vaping can actually improve some health conditions such as tonsilitis [11], that it can reverse symptoms of chronic idiopathic neutrophilia [12], and that it can improve and enhance a sense of well-being [13].

Vaping is typically regarded as a safer alternative to cigarette smoking under the assumption that vaping is less toxic than tobacco use [14–17]. Some researchers are suggesting that adult smokers could vape as a means to reduce or quit smoking tobacco cigarettes [18, 19].

But other studies indicate that vaping can actually lead to nicotine addiction and can have serious negative medical implications [20]. Emerging animal studies are suggesting that vaping may have negative health consequences. For example, an increased susceptibility to infections was found in mice when exposed to vapor [21]. Increased bacterial growth, formation of biofilms, alterations in immunity, and disturbances in airway cytokines were other negative health effects found in animals exposed to vapor [22]. The high temperatures used to heat inhaled vape fluid are leading to the formation of toxins such as acrolein, acetaldehyde, and formaldehyde in the pulmonary system [23, 24]. Alterations in physiological hemostasis, hyperactive platelet activity, and an increased risk for thrombogenic events were also discovered [25].

Human vaping studies are suggesting concerning health outcomes including lung diseases [26], cancer, cardiovascular disorders [27], headaches, bleeding from the nares, weight changes [28, 29], dizziness, nausea, and tremors [30]. Reports of heart attacks and seizures are also emerging as alarming health problems thought to be linked to vape use [31].

Additionally, the US FDA has deemed vaping to be potentially harmful to humans because of the nitrosamines, diethylene glycol, rimonabant, heavy metal, formaldehyde, acetaldehyde, and other contaminants found after using the devices [32]. As such, they have recently implemented new regulations and restrictions for sellers of vape products [5].

Health effects of base solvents in vape fluid

Most vape fluid contains vegetable glycerine and propolyene glycol as the base ingredidents. These FDA approved food ingredients may have concerning health effects when inhaled. Cervellati et al[15] found that base humectants evoked release of cytokines and pro-inflammatory mediators which is concerning even for vapers who do not add nicotine or flavorings to the vape fluid. Other researchers agree citing the discovery that propylene glycol is converted to propylene oxide which can cause a range of health problems ranging from symptoms of infection to carcinogenic effects [26, 27]. Vaporizing vegetable glycerine has been found to cause irritation of the skin, eyes, nares, and esophagus and may also be associated with the development of malignacies [33]. Some studies are finding that the elevated ratios of vegetable glycerin and propylene glycol can lead to reactive oxygen species formation which is linked to cardiovascular, neurodegenerative, sensory, and psychiatric disorders [34]. Other studies suggest that inhaled base ingredients can be converted to dangerous substances when heated. Some of these ingredients include: acetaldehyde, acrolein, acetone, formaldehyde and glyosal. They pose a risk of inducing systemic biological alterations which can lead to inflammation, central nervous system depression, malignancies, and alterations in circadian rhythms [35]. These concerning reports and variants in vape use require much more investigation to fully understand the implications of this new non-tobacco trend.

Gaps in the literature

Very few studies have been completed looking at the physiological effects of vape use or second-hand exposure to vapor despite a call for evidence on that end. A few studies discovered that vaping (with nicotine) increases heart rate (HR) and/or blood pressure (BP) [35, 36]. Vardavas' group [37] determined that vaping led to respiratory impedance and flow resistance. Additionally, more data are needed on the short-term physiological effects of vaping and second-hand inhalation of vapor in order to build up evidence to guide public health practices. The relative recent increase in popularity of vaping coupled with an insufficient amount of research surrounding its effects is affecting the ability for health practitioners to guide treatment.

The identified deficiencies in knowledge about vaping and the need to contribute knowledge to inform health providers about the safety and health effects of vape use prompted the physiological variables of HR, BP, respiratory rate (RR), blood sugar (BS), oxygen saturation (O_2%) and pulmonary function tests (PFT) to be chosen for examination in this study. Additionally, we were interested in understanding the effects of

second-hand vapor for people exposed to vapor but not actually using the vape as well. Finally, we attempted to identify demographic differences between the two groups.

Purpose and hypothesis

The purpose of this study was to examine the physiological effects of vaping and second-hand exposure to vapor when vaping for twenty minutes without nicotine or added flavorings. It was expected that both direct inhalation of vegetable glycerine and propylene glycol vapor through a vape device, and inhalation of second-hand vapor by those not using a vape device but exposed to such vapor would contribute to an increase in HR, BP (systolic, diastolic, and mean arterial pressure), respiratory rate (RR), and blood sugar (BS). We also hypothesized that there would be a decrease in percent of blood oxygen saturation and pulmonary function test (PFT) results.

Materials and methods

Design and procedure

The study utilized a mixed-factorial experimental design with one "between groups" factor (i.e., participants who directly vaped versus participants who did not directly vape but were exposed to second-hand vapor), and eight repeated measures of 'within subjects' factors (i.e., measurements of each of the eight physiological variables both before and after vaping or exposure to second hand vapor). Institutional Review Board (IRB) approval was obtained from the institution of the first author (i.e., University of Blinded for Review) prior to the commencement of the study.

Participants from both the vape and non-vape groups were commingled in data gathering sessions such that non-vape participants were sat next to vape participants. For each session, participants first provided informed consent and then were asked to complete a demographic and health history questionnaire. Thereafter, all physiological variables were measured. Next, vaping participants were given a Sorin® vape device filled with a 70/30 mix of vegetable glycerin and propylene glycol and asked to vape for twenty minutes. The fluid contained no nicotine or flavorings in order to determine the physiological effects of the base vape fluid without additives. Non-vapers were asked to sit next to the vaping participants during the same time frame. At the conclusion of the twenty minutes of vaping, physiological measurements were taken again.

Sample

A convenience sample of adult volunteers was solicited using social media and direct recruitment by the researchers. A total of 148 volunteers agreed to participate. Approximately half of the participants self-identified as vape users and were assigned to the vape group, while the remaining participants self-identified as non-vape users and were assigned to the non-vape (i.e., vape-exposure) group. Participants were asked to not eat or vape at least sixty minutes prior to data collection.

Measures

Physiological variables measured included: HR measured in beats per minute (bpm), respiratory rate (RR) measured in breaths per minute, percent of blood oxygen saturation (% O_2 sat) measured using a pulse oximeter, BP measured using manual blood pressure cuff with ausculation for systolic (SBP) over diastolic (DBP) pressures (mm Hg), mean arterial pressure (MAP) determined by calculating SBP + 2(DBP)/3, pulmonary function tests (PFT) measured using a peak flow meter (% of personal best average of three attempts), and blood sugar (BS) measured using a glucometer (mg/dL).

Results

Demographic characteristics and participant responses to all health variables for the entire sample are reported separately for each experimental group (i.e., non-vape and vape) in Table 1. Analyses that were completed to determine if the two groups differed on any of these variables are also reported in the table. Of these analyses, only those involving gender and smoking habits emerged significant. The finding for smoking habits became non-significant when participants identified as former smokers were removed from the analysis. It was also interesting to note that while the two groups did not demonstrate statistically significant differences, a higher frequency of vapers reported health risk factors including alcoholism, use of alcohol, cigarette use, former cigarette use, and mental illness compared to the non-vaping participants. Conversely, participants in the non-vaping group reported a higher frequency of a positive family history for alcoholism.

Basic descriptive statistics were calculated for age and all outcome variables of interest for the entire sample, as well as for the non-vape and

Table 1. Descriptive statistics and frequencies for all demographic and health variables for the pooled sample and the non-vape and vape groups separately

	Total sample (N = 148)	Non-vape group (n = 75)	Vape group (n = 73)	Tests of group differences
Age				
Mean	23.19	22.64	23.76	F (1,136) = 0.76, p = 0.386
Standard deviation	9.23	8.08	10.30	
Minimum–maximum	18–78	18–78	18–62	
Gender				
Male	63	22	41	$\chi^2(1) = 10.30$, p = 0.001
Female	79	49	30	
Missing	6	4	2	
Race				
White	112	60	52	$\chi^2(1) = 3.36$, p = 0.067
Not white/mixed	29	10	19	
Missing	7	5	2	
Family history of alcoholism				
No	118	58	60	$\chi^2(1) = 0.34$, p = 0.561
Yes	29	16	13	
Unsure/missing	1	1	0	
Alcoholism				
No	142	73	69	$\chi^2(1) = 2.09$, p = 0.149
Yes	2	0	2	
Unsure/missing	4	2	2	
Uses alcohol				
No	42	26	16	$\chi^2(1) = 2.98$, p = 0.084
Yes	102	47	55	
Missing	4	2	2	
Smoking habits				
Non-smoker	133	72	61	$\chi^2(2) = 8.55$, p = 0.014
Smoker	6	1	5	
Former smoker	5	0	5	
Missing	4	2	2	
Drug addiction				
No	139	70	69	$\chi^2(1) = 0.00$, p = 0.989
Yes	4	2	2	
Unsure/missing	5	3	2	
Mental illness				
No	111	58	53	$\chi^2(1) = 0.49$, p = 0.485
Yes	31	14	17	
Unsure/missing	6	3	3	

For age, four cases were missing data, so calculations based on N = 144 for the pooled sample, n = 73 for the non-vape group, and n = 71 for the vape group, respectively. For tests of group differences, one-way analysis of variance was used with age, and chi-square tests of independence were used for all other variables. For smoking habits, the chi-square analysis was run a second time after excluding former smokers. The result emerged non-significant; $\chi^2(1) = 3.23$, p =0.072

vape groups separately. As well, skew and kurtosis were computed for all variables. An examination of these statistics revealed evidence of a severe non-normality of score distributions for multiple variables as reflected in the elevated skew (i.e., values of 2 or greater) and/or kurtosis (i.e., values of 7 or greater). In addition, most variables appear to have extreme values (i.e., values that are three standard deviations below or above the mean).

Study outcomes as a function of demographic and health variables

Prior to running the main analyses, we thought it would be worthwhile to examine the extent to which all main study outcome variables may be influenced by demographic and health variables. As such, we computed product-moment correlations with age and outcome variables via one-way Analysis of Variance (ANOVA) wherein the outcome variables served as dependent variables, and the remaining demographic (e.g., gender, race) and health variables (i.e., smoking habit, family history of alcoholism, alcoholism, use of alcohol, addiction, and mental illness) were used as independent variables. While not reported in this article due to length restrictions, one or more statistically significant results was obtained with all variables except self-reported alcoholism. The most frequent significant results were found for gender, followed by age, addiction, family history of alcoholism, alcohol use, race, mental illness, and smoking habit, respectively.

Main analyses

Given the research design used in this study, the most straightforward and efficient approach to analysis would have involved the completion of eight mixed factorial ANOVAs with the groups (i.e., non-vape vs. vape) as the "between groups" independent variable, and the pre-post measurements of the outcome variables (i.e., pre-post systolic blood pressure, diastolic blood pressure, mean arterial pressure, heart rate, blood oxygen saturation, respiratory rate, blood sugar, and pulmonary functions) as the 'within subjects' variables. However, problems with the non-normality of score distributions and/or extreme outlying values of many variables challenged the appropriateness of using such a statistic because it is known to be sensitive to violations of assumptions regarding normality, and also because extreme values have a distorting influence on mean scores.

In lieu of the mixed factorial ANOVAs, we decided to adopt a multi-staged approach to data analysis. First, we completed one-way ANOVAs using each outcome variable as the dependent variable and experimental groups as the independent variable. Second, we re-ran ANOVAs after excluding extreme values on the dependent variable whenever extreme outliers were found. This was done to ensure that results were not skewed due to the influence of outliers. Third, we ran Mann-Whitney tests using all data including outliers. This is the non-parametric equivalent of the one-way ANOVA and is not influenced by non-normality. The results of these three sets of analyses using pre-test, post-test, and pre-post difference variables can be found in Tables 2 and 3, respectively.

An examination of the results for the pre-test variables in Table 2 revealed significant findings in all analyses for systolic blood pressure, mean arterial pressure, and pulmonary functions with the vape group obtaining the significantly higher mean score in all cases. Effect sizes as reflected in eta-squared are all small. In addition, while the one-way ANOVA for blood oxygen saturation was found to be non-significant in all the cases, the result became significant when excluding outliers. The Mann-Whitney test result was also significant. The effect size, however, is small. The non-vape group obtained the higher mean score.

In terms of the post-test variables in Table 2, consistently significant results with small effect sizes were obtained for systolic blood pressure, diastolic blood pressure, mean arterial pressure, and pulmonary function tests. In all cases, the vape group produced the higher mean score. Alternatively, an inspection of Table 3 shows that no significant results were found with the pre-post difference scores. This indicates that the degree of change in values from pre- to post-test was not markedly different across the non-vape and vape groups for any of the measured outcome variables.

To further test for the robustness of results, two additional sets of analyses were completed. In the first, outliers on both the dependent variable and age were excluded and one-way ANOVAs were re-computed. In the second, with the same outliers removed, we completed Analyses of Covariance (ANCOVA) controlling for age and gender. These variables were selected for use as covariates based upon the large number of statistically significant findings that we obtained when examining their association with the outcome variables.

Table 2. Results of analyses examining all pre-test and post-test physiological variables as a function of experimental group (non-vape *vs* vape)

	Non-vape (n = 75)	Vape (n = 73)	One-way ANOVA			One-way ANOVA with outliers excluded			Mann-Whitney test	
	Mean [sd]	Mean [sd]	F (1,146)	P	Eta^2	F [df_n/df_d]	P	Eta^2	U (z)	P
Pre-test physiological measurements										
Systolic blood pressure	119.81 (14.52)	125.93 (15.45)	6.17	0.014	0.04	5.24 (1, 145)	0.024	0.03	2093.00 (-2.47)	0.013
Diastolic blood pressure	74.93 (8.18)	77.70 (9.73)	3.51	0.063		—	—	—	2284.00 (-1.74)	0.081
Mean arterial pressure	89.89 (8.97)	93.78 (10.73)	5.71	0.018	0.04	4.78 (1, 145)	0.030	0.03	2179.00 (-2.14)	0.032
Heart rate	85.61 (16.39)	86.62 (17.90)	0.13	0.723		0.13 (1, 144)	0.720		2728.50 (-0.03)	0.972
Blood O_2 saturation	98.16 (1.17)	97.77 (1.30)	3.74	0.055		4.45 (1, 143)	0.037	0.03	2179.50 (-2.27)	0.024
Respiratory rate	11.84 (1.67)	12.05 (1.63)	0.63	0.430		0.91 (1, 144)	0.342		2528.50 (-0.93)	0.351
Blood sugar	99.09 (18.43)	98.82 (23.58)	0.01	0.938		0.50 (1, 143)	0.483		2528.50 (-0.80)	0.423
Pulmonary function test	405.80 (111.43)	458.36 (124.88)	7.31	0.008	0.05	9.48 (1, 145)	0.003	0.06	2047.00 (-2.65)	0.008
Post-test physiological measurements										
Systolic blood pressure	115.37 (13.85)	120.78 (13.66)	5.71	0.018	0.04	4.83 (1, 145)	0.030	0.03	2127.50 (-2.34)	0.019
Diastolic blood pressure	72.37 (8.06)	77.00 (10.77)	8.79*	0.004	0.06	7.72* (1, 145)	0.006	0.05	1992.00 (-2.87)	0.004
Mean arterial pressure	86.71 (8.69)	91.59 (10.79)	9.23*	0.003	0.06	—	—	—	1992.00 (-2.86)	0.004
Heart rate	83.04 (14.00)	82.77 (16.43)	0.01*	0.913		0.02* (1, 145)	0.882		2713.00 (-0.09)	0.925
Blood O_2 saturation	97.93 (1.28)	97.74 (1.14)	0.94	0.333		1.62 (1, 143)	0.205		2384.00 (-1.44)	0.149
Respiratory rate	12.36 (2.50)	12.19 (1.60)	0.24	0.628		0.20 (1, 144)	0.655		2642.50 (-0.40)	0.690
Blood sugar	99.35 (17.73)	98.93 (21.93)	0.02	0.899		0.08 (1, 144)	0.779		2666.50 (-0.27)	0.785
Pulmonary function test	403.05 (124.16)	455.18 (137.73)	5.85	0.017	0.04	—	—	—	2046.00 (-2.65)	0.008

One-way ANOVAs with Outliers Excluded were only computed when there was evidence of one or more extreme outliers (i.e., values greater than 3 standard deviations from the total pooled mean) on the dependent variable. mean arterial pressure was estimated using the formula [systolic blood press + (2 × diastolic blood press)] / 3. Asterisk (*) means that Levene's test was significant so homogeneity of variance cannot be assumed

Table 3. Results of analyses examining all pre-post test difference scores as a function of experimental group (non-vape *vs* vape)

	Non-vape (n = 75)	Vape (n = 73)	One-way ANOVA			One-way ANOVA with outliers excluded			Mann-Whitney test	
	Mean [sd]	Mean [sd]	F (1,146)	P	Eta^2	F [df_n/df_d]	P	Eta^2	U (z)	P
Systolic blood pressure	4.44 (10.34)	5.15 (12.14)	0.15	0.702		0.55 (1, 145)	0.460		2612.50 (-0.48)	0.631
Diastolic blood pressure	2.56 (8.34)	0.70 (7.99)	1.92	0.168		—	—	—	2348.50 (-1.50)	0.135
Mean arterial pressure	3.19 (7.70)	2.18 (7.90)	0.61	0.435		—	—	—	2599.00	0.595
Heart rate	2.57 (14.30)	3.85 (14.08)	0.30	0.585		0.04 (1, 145)	0.851		2687.00 (-0.58)	0.564
Blood O_2 saturation	0.23 (1.44)	0.03 (1.47)	0.69	0.406		0.04 (1, 142)	0.840		2530.00 (-0.83)	0.404
Respiratory rate	-0.52 (2.81)	-0.14 (2.04)	0.90	0.345		0.02 (1, 143)	0.902		2688.00 (-0.20)	0.843
Blood sugar	-0.25 (16.99)	-0.11 (16.90)	0.00	0.959		0.03 (1, 144)	0.867		2674.50 (-0.24)	0.809
Pulmonary function test	2.75 (42.37)	3.18 (52.21)	0.00	0.956		0.44 (1, 144)	0.507		2523.00 (-0.82)	0.410

Pre-post difference scores were computed by subtracting post-test values from pre-test values for each variable. One-way ANOVAs with Outliers Excluded were only computed when there was evidence of one or more extreme outliers (i.e., values greater than 3 standard deviations from the total pooled mean) on the dependent variable. Mean arterial pressure was estimated using the formula [systolic blood press + (2 × diastolic blood press)] / 3. Asterisk (*) means that Levene's test was significant so homogeneity of variance cannot be assumed

Analyses that emerged non-significant in the first three sets of analyses continued to be non-significant in these additional analyses. The significant findings found with pre-test systolic blood pressure, pre-test mean arterial pressure, pre-test blood oxygen saturation, post-test systolic blood pressure, and post-test pulmonary functioning came out non-significant after excluding outliers on these variables and age and when controlling for age and gender. Pre-test pulmonary function, post-test diastolic blood pressure, and post-test mean arterial pressure were found to remain significant after excluding outliers on the variables as well as age but became non-significant in the ANCOVAs. Overall, it appears that all significant "between-group" results can be seen as a product of the influence of outlier scores and/or covariates.

To provide a more fulsome evaluation of the effects of vape exposure on the non-vape and vape groups, we elected to complete a number of repeated measures ANOVAs for each group separately. Akin to the between-groups analyses, we used a multi-staged approach where we first examined the pairs of pre-post outcome variables using all available data for each group. Thereafter, we ran a second set of ANOVAs excluding outliers on both the pre- and/or post-test outcome variables. Third, we ran Wilcoxon tests for each pre-post variable pair. This statistic is the nonparametric equivalent to a paired-samples t-test and repeated measures ANOVA and is not influenced by non-normality or extreme outliers. Results of these analyses can be found in Table 4.

Inspection of the findings for the non-vape group only showed significant results with small to medium effect sizes for systolic blood pressure, diastolic blood pressure, and mean arterial pressure. In all instances, pre-test mean scores were significantly higher. No other significant results were found for the non-vape group. For the vape group, systolic blood pressure, mean arterial pressure, and heart rate (all higher at pre-test) came out significant with small to medium effect sizes.

Lastly, we ran two additional sets of repeated measures ANOVAs. In one set, outlier pre- and/or post-test scores on the outcome variable were excluded, as were outliers on age. In the second, outliers continued to be excluded and both gender and age were used as covariates. Results were mostly the same as they were in the first three sets of analyses. For the non-vape group, systolic blood pressure, diastolic blood pressure, and mean arterial pressure remained statistically significant with small-to-medium effect sizes. For the vape group, systolic blood pressure remained

Table 4. Results of analyses examining all dependent variables at pre-post test for experimental groups separately

	Pre-test	Post-test	Repeated measures ANOVA			Repeated measures ANOVA with outliers excluded			Wilcoxon test	
	Mean [SD]	Mean [SD]	F [df_n/df_d]	P	Partial eta^2	F [df_n/df_d]	P	Partial eta^2	Z	P
Non-vape group only (n = 75)										
Systolic blood pressure	119.81 (14.52)	115.37 (13.85)	13.82 (1, 74)	< 0.001	0.16	—	—	—	-3.39	<0.001
Diastolic blood pressure	74.93 (8.18)	72.37 (8.06)	7.07 (1, 74)	0.010	0.09	—	—	—	-2.65	0.008
Mean arterial pressure	89.89 (8.97)	86.71 (8.69)	12.86	0.001	0.15	—	—	—	-3.15	0.002
Heart rate	85.61 (16.39)	83.04 (14.00)	2.43 (1, 74)	0.124		2.23 (1, 73)	0.139		-1.22	0.223
Blood O_2 saturation	98.16 (1.17)	97.93 (1.28)	1.86 (1, 74)	0.177		3.52 (1, 72)	0.065		-1.62	0.105
Respiratory rate	11.84 (1.67)	12.36 (2.50)	2.57 (1, 74)	0.113		1.24 (1, 71)	0.270		-1.07	0.285
Blood sugar	99.09 (18.43)	99.35 (17.73)	0.02 (1, 74)	0.898		0.04 (1, 71)	0.849		-0.03	0.975
Pulmonary function test	405.80 (111.43)	403.05 (124.16)	0.32 (1, 74)	0.576		1.17 (1, 71)	0.284		-1.25	0.212
Vape group only (n =73)										
Systolic blood pressure	125.93 (15.45)	120.78 (13.66)	13.14 (1, 72)	0.001	0.15	—	—	—	-3.58	<0.001
Diastolic blood pressure	77.70 (9.73)	77.00 (10.77)	0.56 (1, 72)	0.457		—	—	—	-0.80	0.423
Mean arterial pressure	93.78 (10.73)	91.59 (10.79)	5.57	0.021	0.07	—	—	—	-2.43	0.015
Heart rate	86.62 (17.90)	82.77 (16.43)	5.45 (1, 72)	0.022	0.07	4.49 (1, 71)	0.038	0.06	-2.23	0.026
Blood O_2 saturation	97.77 (1.30)	97.74 (1.14)	0.03 (1, 72)	0.874		0.35 (1, 69)	0.556		-0.83	0.407
Respiratory rate	12.05 (1.63)	12.19 (1.60)	0.33 (1, 72)	0.567		0.58 (1, 70)	0.450		-0.56	0.579
Blood sugar	99.82 (23.58)	98.93 (21.93)	0.00 (1, 72)	0.956		0.06 (1, 71)	0.806		-0.28	0.781
Pulmonary function test	458.36 (124.88)	455.18 (137.73)	0.27 (1, 72)	0.605		—	—	—	-0.21	0.833

Repeated measures ANOVAs with outliers excluded were only computed when there was evidence of one or more extreme outliers (i.e., values greater than 3 standard deviations from the group mean) on the dependent variable at pre- and/or post-test. Mean arterial pressure was estimated using the formula [systolic blood press + (2 × diastolic blood press)] / 3

significant in both sets of analyses with medium effect sizes. Mean arterial pressure continued to be significant when outliers were removed but dropped below statistical significance when covariates were included. Finally, heart rate emerged non-significant in both analyses.

Discussion

The findings of this investigation provide an interesting and somewhat unexpected set of results. In relation to our hypothesis which predicted that HR, BP, respiratory rate (RR), and blood sugar (BS) would increase while the percent of blood oxygen saturation (O_2%) and results of pulmonary function testing (PFT) would decrease as a function of vaping and/or exposure to second-hand vapor, our pre-post analyses indicated that, for the vape group, SBP, mean arterial pressure (MAP), and HR significantly decreased after vaping. However, only the result for SBP remained significant when controlling

for age and gender and/or excluding outliers. For the non-vape group, all three BP variables (SBP, DBP, and MAP) were significantly lower after exposure to second-hand vapor. When considering our 'between-groups' analyses, the vape group produced significantly higher pre- and post-vaping mean values on SBP, MAP, and PFTs while obtaining a significantly higher mean DBP at post-vape only. Nevertheless, all of these differences ceased being statistically significant when excluding outliers and/or when controlling for age and gender. As well, "between-group" analyses of mean pre-post difference scores were consistently non-significant indicating that the two groups did not substantively differ from each other in their physiological response to vaping/exposure to vapor. Based upon our findings, it appears that vegetable glycerine and propylene glycol e-juice without flavoring or nicotine does not seem to have any markedly negative immediate physiological effects regardless of whether they were directly vaped or inhaled through second-hand vapor.

The fact that robust decreases in one or more mean BP variables were found for both groups after vaping or exposure to vapor is counterintuitive but may be explained as a function of all participants becoming more relaxed with the experiment as participation in the study proceeded (e.g., participants in both groups became more comfortable with the experimental situation and interacting with each other). If this is correct, then it may be worthwhile for future researchers using a similar methodology to ensure that participants are at ease with each other and with the study conditions prior to vaping or exposing participants to vapor. It might also be good to obtain two or more baseline (i.e., before vaping or exposure to vapor) physiological measurements so that the effects of any initial discomfort can be mitigated through the averaging of multiple measurements.

Demographic findings suggest that people who chose to vape show a trend toward reporting higher levels of other health-risk behaviors including abusing alcohol and cigarette use. Vapers also reported a higher frequency of drinking alcohol and have a higher incidence of mental illness compared to people who do not vape. Conversely, people who do not vape reported a higher incidence of a positive family history of alcoholism. However, the absence of statistically significant differences between the groups on most of these variables (i.e all but smoking) makes it difficult to ascertain if the higher levels of health-risk behaviors seen with our sample are reflective of robust differences that would be seen in the population at large.

Limitations of the study

The study was limited by the skewed gender and race distribution in the sample. There were significantly more females than males and a disproportionately high percentage of Caucasian participants compared to other ethnic groups. A more equitably diverse sample may yield different results. Additionally, the age of participants was established as needing to be greater than 18. However, there was no upper limit and since age can affect some vital signs, similar future studies could consider capping the upper age limit.

Conclusions

One of the main goals of medical professionals is to promote health and reduce risk. It is currently unclear if long-term vape use is a safe alternative to smoking or if the use of e-cigarettes can help smokers quit smoking [38, 39]. The outbreak of lung diseases portrayed in many media outlets during summer 2019 created public awareness and concern regarding the health effects of vape use. Policy makers have been implementing regulations on vaping in an attempt to keep the public safe, but this is often done with insufficient evidence [40]. Medical providers need to stay abreast of current vaping research in order to know how to best guide patients, the public at large, and contribute to evidenced based laws, policies, and regulations surrounding vape use.

Conflict of interest

None declared.

References:

1. Levy DT, Cummings KM, Villanti AC, et al. A framework for evaluating the public health impact of e-cigarettes and other vaporized nicotine products. Addiction. 2017; 112(1): 8–17, doi: 10.1111/add.13394, indexed in Pubmed: 27109256.
2. Rimer S. Chasing the facts about e-cigarette health risks. Boston University Research. Behind the Vapor, July 11, 2017. Available online: www.bu.edu/eng/2016/05/12/chasing-the-facts-about-e-cigarette-health-risks/. [Last accessed at: 05.09.2019].
3. Center for Disease Control. Chapter 1 Introduction, Conclusions, and Historical Background Relative to E-Cigarettes. In S. General, E-Cigarette Use Among Youth and Young Adults (pp. 1-24). Washington, DC: CDC.gov. Available online: www.cdc.gov/tobacco/data_statistics/sgr/e-cigarettes/pdfs/2016_SGR_Chap_1_508.pdf. [Last accessed at: 05.09.2019].
4. Nurasyikin MS, Leelavathi M, Tohid H. E-Cigarette use, its impact on tobacco smoking. Med & Health. 2019; 14(1): 78–90, doi: https://doi.org/10.17576/MH.2019.1401.07.

5. Cai K. Juul and rivals, given 10 months to submit FDA application, face battle to keep selling e-cigs. Forbes. 16.07.2019. Available online: https://www.forbes.com/sites/kenrickcai/2019/07/16/juul-rivals-10-month-deadline-fda-application-battle-to-sell-e-cigarettes/#5cd160361f3c. [Last accessed at: 20.09.2019].
6. Food and Drug Administration, HHS. Deeming tobacco products to be subject to the federal food, drug, and cosmetic act, as amended by the family smoking prevention and tobacco control act; restrictions on the sale and distribution of tobacco products and required warning statements for tobacco products. Final rule. Fed Regist. 2016; 81(90): 28973–29106, indexed in Pubmed: 27192730.
7. Martin EM, Clapp PW, Rebuli ME, et al. E-cigarette use results in suppression of immune and inflammatory-response genes in nasal epithelial cells similar to cigarette smoke. Am J Physiol Lung Cell Mol Physiol. 2016; 311(1): L135–L144, doi: 10.1152/ajplung.00170.2016, indexed in Pubmed: 27288488.
8. Pepper JK, Brewer NT. Electronic nicotine delivery system (electronic cigarette) awareness, use, reactions and beliefs: a systematic review. Tob Control. 2014; 23(5): 375–384, doi: 10.1136/tobaccocontrol-2013-051122, indexed in Pubmed: 24259045.
9. Polosa R, Caponnetto P, Cibella F, et al. Quit and smoking reduction rates in vape shop consumers: a prospective 12-month survey. Int J Environ Res Public Health. 2015; 12(4): 3428–3438, doi: 10.3390/ijerph120403428, indexed in Pubmed: 25811767.
10. Stead L, Perera R, Bullen C, et al. Nicotine replacement therapy for smoking cessation. Cochrane Database of Systematic Reviews. 2012, doi: 10.1002/14651858.cd000146.pub4.
11. Miler JA, Hajek P. Resolution of recurrent tonsillitis in a non-smoker who became a vaper. A case study and new hypothesis. Med Hypotheses. 2017; 109: 17–18, doi: 10.1016/j.mehy.2017.09.006, indexed in Pubmed: 29150279.
12. Farsalinos KE, Romagna G. Chronic idiopathic neutrophilia in a smoker, relieved after smoking cessation with the use of electronic cigarette: a case report. Clin Med Insights Case Rep. 2013; 6: 15–21, doi: 10.4137/CCRep.S11175, indexed in Pubmed: 23439796.
13. Keane H, Weier M, Fraser D, et al. 'Anytime, anywhere': vaping as social practice. Critical Public Health. 2016; 27(4): 465–476, doi: 10.1080/09581596.2016.1250867.
14. Carter B, Clark A, Woessner J. CASAA submits comment on FDA's smokeless tobacco standards. Consumer Advocates for Smoke Free Alternatives Association. Washington, D.C. Available online: www.casaa.org/news/casaa-submits-comment-on-fdas-smokeless-tobacco-standards-rule/. [Last accessed at: 30.09.2019].
15. Cervellati F, Muresan XM, Sticozzi C, et al. Comparative effects between electronic and cigarette smoke in human keratinocytes and epithelial lung cells. Toxicol In Vitro. 2014; 28(5): 999–1005, doi: 10.1016/j.tiv.2014.04.012, indexed in Pubmed: 24809892.
16. Chedekel L, Rimer S. Public Health professor Michael Siegel says concerns are premature and overblown. Behind the Vapor. Available online: www.bu.edu/research/articles/behind-the-vapor/. [Last accessed at: 24.09.2019].
17. McNeill A, Brose LS, Calder R, et al. E-cigarettes: an evidence updateA report commissioned by Public Health England 2015. Available online: https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/733022/Ecigarettes_an_evidence_update_A_report_commissioned_by_Public_Health_England_FINAL.pdf. [Last accessed at: 30.09.2019].
18. Palmer A. Parsing the influences of nicotine and expectancies on the acute effects of e-cigarettes: A balanced-placebo experiment. Graduate Theses and Dissertations. Tampa: Clinical Psychology Commons. Available online: http://scholarcommons.usf.edu/etd/6923. [Last accessed at: 01.10.2019].
19. Palmer AM, Brandon TH. Nicotine or expectancies? Using the balanced-placebo design to test the immediate outcomes of vaping. *Addictive Behaviors.* 2019; 97: 90-96. doi: 10.1016/j.addbeh.2019.04.026.
20. Hua My, Alfi M, Talbot P. Health-related effects reported by electronic cigarette users in online forums. J Med Internet Res. 2013; 15(4): e59, doi: 10.2196/jmir.2324, indexed in Pubmed: 23567935.
21. Sussan TE, Gajghate S, Thimmulappa RK, et al. Exposure to electronic cigarettes impairs pulmonary anti-bacterial and anti-viral defenses in a mouse model. PLoS One. 2015; 10(2): e0116861, doi: 10.1371/journal.pone.0116861, indexed in Pubmed: 25651083.
22. Hwang JH, Lyes M, Sladewski K, et al. Electronic cigarette inhalation alters innate immunity and airway cytokines while increasing the virulence of colonizing bacteria. J Mol Med (Berl). 2016; 94(6): 667–679, doi: 10.1007/s00109-016-1378-3, indexed in Pubmed: 26804311.
23. Geiss O, Bianchi I, Barrero-Moreno J. Correlation of volatile carbonyl yields emitted by e-cigarettes with the temperature of the heating coil and the perceived sensorial quality of the generated vapours. Int J Hyg Environ Health. 2016; 219(3): 268–277, doi: 10.1016/j.ijheh.2016.01.004, indexed in Pubmed: 26847410.
24. Gillman IG, Kistler KA, Stewart EW, et al. Effect of variable power levels on the yield of total aerosol mass and formation of aldehydes in e-cigarette aerosols. Regul Toxicol Pharmacol. 2016; 75: 58–65, doi: 10.1016/j.yrtph.2015.12.019, indexed in Pubmed: 26743740.
25. Qasim H, Karim ZA, Silva-Espinoza JC, et al. Short-Term e-cigarette exposure increases the risk of thrombogenesis and enhances platelet function in mice. J Am Heart Assoc. 2018; 7(15), doi: 10.1161/JAHA.118.009264, indexed in Pubmed: 30021806.
26. Eltorai AEm, Choi AR, Eltorai AS. Impact of electronic cigarettes on various organ systems. Respir Care. 2019; 64(3): 328–336, doi: 10.4187/respcare.06300, indexed in Pubmed: 30401756.
27. Glantz SA, Bareham DW. E-Cigarettes: use, effects on smoking, risks, and policy implications. Annu Rev Public Health. 2018; 39: 215–235, doi: 10.1146/annurev-publhealth-040617-013757, indexed in Pubmed: 29323609.
28. Centers for Disease Control and Prevention. New CDC study finds dramatic increase in e-cigarette-related calls to poison centers. Available online: www.cdc.gov/media/releases/2014/p0403-e-cigarette-poison.html. [Last accessed at: 15.09.2019].
29. Lanza HI, Pittman P, Batshoun J. Obesity and cigarette smoking: extending the link to e-cigarette/vaping use. Am J Health Behav. 2017; 41(3): 338–347, doi: 10.5993/AJHB.41.3.13, indexed in Pubmed: 28376978.
30. Voytko LF. Investigates 127 Seizure Reports Potentially Linked To Vaping. New York: Forbes. August 10, 2019. Available online: www.forbes.com/sites/lisettevoytko/2019/08/08/fda-investigates-127-seizure-reports-potentially-linked-to-vaping/#4997f442454b. [Last accessed at: 05.09.2019].
31. Alzahrani T, Pena I, Temesgen N, et al. Association between electronic cigarette use and myocardial infarction. Am J Prev Med. 2018; 55(4): 455–461, doi: 10.1016/j.amepre.2018.05.004, indexed in Pubmed: 30166079.
32. Gottlieb S, Abernethy A. Understanding the Health Impact and Dangers of Smoke and 'Vapor'. St. Louis: FDA News room. Food and Drug Administration. Available online: www.fda.gov/news-events/fda-voices-perspectives-fda-leadership-and-experts/understanding-health-impact-and-dangers-smoke-and-vapor. [Last accessed at: 05.09.2019].
33. Ween M, Hodge G, Reynolds P. A26 Smoke, pollutants, and toxins: Insights from bench studies: The new kid on the block: E-cigarettes can cause damage to airway cells and cause airway macrophage dysfunction. Am J Resp and Critical Care Medicine. 2016; 193.
34. Brieger K, Schiavone S, Miller FJ, et al. Reactive oxygen species: from health to disease. Swiss Med Wkly. 2012; 142: w13659, doi: 10.4414/smw.2012.13659, indexed in Pubmed: 22903797.
35. Lechasseur A, Jubinville É, Routhier J, et al. Exposure to electronic cigarette vapors affects pulmonary and systemic expression of circadian molecular clock genes. Physiol Rep. 2017; 5(19), doi: 10.14814/phy2.13440, indexed in Pubmed: 29038357.
36. Vlachopoulos C, Ioakeimidis N, Abdelrasoul M, et al. Electronic cigarette smoking increases aortic stiffness and blood pressure

in young smokers. J Am Coll Cardiol. 2016; 67(23): 2802–2803, doi: 10.1016/j.jacc.2016.03.569, indexed in Pubmed: 27282901.
37. Vardavas CI, Anagnostopoulos N, Kougias M, et al. Short-term pulmonary effects of using an electronic cigarette: impact on respiratory flow resistance, impedance, and exhaled nitric oxide. Chest. 2012; 141(6): 1400–1406, doi: 10.1378/chest.11-2443, indexed in Pubmed: 22194587.
38. Ashford K, Rayens E, Wiggins AT, et al. Advertising exposure and use of e-cigarettes among female current and former tobacco users of childbearing age. Public Health Nurs. 2017; 34(5): 430–436, doi: 10.1111/phn.12334, indexed in Pubmed: 28485553.
39. English TM, Smith TB, Song X, et al. Barriers to electronic cigarette use. Public Health Nurs. 2018; 35(5): 363–368, doi: 10.1111/phn.12406, indexed in Pubmed: 29984426.
40. LaVito A. Michigan becomes first state to ban sales of flavored e-cigarettes. [CNBC]. Available online: https://www.cnbc.com/2019/09/04/michigan-bans-sales-of-flavored-e-cigarettes-restricts-vaping-marketing.html. [Last accessed: 05.09.2019].