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Psychological and cognitive effects of laser printer emissions: A controlled exposure study

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Abstract

The possible impact of ultrafine particles from laser printers on human health is controversially discussed although there are persons reporting substantial symptoms in relation to these emissions. A randomized, single-blinded, cross-over experimental design with two exposure conditions (high-level and low-level exposure) was conducted with 23 healthy subjects, 14 subjects with mild asthma, and 15 persons reporting symptoms associated with laser printer emissions. To separate physiological and psychological effects, a secondary physiologically based categorization of susceptibility to particle effects was used. In line with results from physiological and biochemical assessments, we found no coherent, differential, or clinically relevant effects of different exposure conditions on subjective complaints and cognitive performance in terms of attention, short-term memory, and psychomotor performance. However, results regarding the psychological characteristics of participants and their situational perception confirm differences between the participants groups: Subjects reporting symptoms associated with laser printer emissions showed a higher psychological susceptibility for adverse reactions in line with previous results on persons with multiple chemical sensitivity or idiopathic environmental intolerance. In conclusion, acute psychological and cognitive effects of laser printer emissions were small and could be attributed only to different participant groups but not to differences in exposure conditions in terms of particle number concentrations.

KEYWORDS

cognitive performance, exposure, idiopathic environmental intolerance, laser printer emission, multiple chemical sensitivity, subjective complaints

1 | INTRODUCTION

Ultrafine particles (UFP) are emitted from laser printers in varying quantities during operation. Their possible impact on human health is controversially discussed, and there are persons reporting substantial symptoms supposedly caused by laser printer emissions. There is some evidence for the biological effectiveness of UFP, particularly from epidemiological studies on outdoor air pollution.[1,2] Fine and ultrafine indoor air particles are therefore seen as a potential health hazard.[3-5] Among these indoor particles, the potential effects of laser printer emissions are not yet sufficiently investigated. Most studies focused on toner powder and used in vitro studies or animal experiments with concentrations often far above the amount a person could take up by inhalation.[6,7] A more relevant health hazard could emanate from particles and volatile organic compounds (VOC) generated and released during the printing process. Several case studies of variable quality described clinical responses after laser printer exposure, such as

cough, headache, or an increased number of eosinophilic granulocytes.[8-10] A study conducted in a copy shop showed an increase in 8-hydroxydesoxyguanosin (8-OhdG) in urine as marker of oxidative stress as well as an increase in pro-inflammatory cytokines in nasal secretions;[11] however, the exposure conditions were not controlled and varied greatly. In cell cultures, the emitted particles or toner powder induced an increase in pro-inflammatory cytokines,[12,13] but such results are difficult to transfer to human subjects as whole organisms. A recent review[14] summarizes this research since the early 2000s. It also shows that standardized experimental studies in a realistic setting with human subjects are still missing. We therefore conducted a controlled exposure study with high and low emitting laser printers and included healthy persons but also subjects who might be regarded as particularly sensitive based on clinical history or physiological considerations.[15] Examinations before and after exposures included lung function measurements and the analysis of biomarkers in serum and nasal secretions. Overall, some small changes were detected but these did not differ between low-level exposure (LLE) and high-level exposure (HLE), and the results did not show a coherent pattern of responses in any of the groups studied (for details, see Karrasch et al[15]). However, although there were no clear-cut physiological responses, psychological or cognitive aspects might still be affected.

There are two related problems when researching the consequences of environmental hazards linked to perceivable sensations like odor or noise. Firstly, as Mølhave[16] states, not for every subjective bodily symptom and its processed evaluation by the reporting person are there objective measures or indicators. For example, a reported malaise usually corresponds to nonspecific symptoms that are not helpful in determining definite causes. Secondly, the sensations themselves might act as markers or stressors.[17] Marker means that the sensation itself, especially of odor, might unfold a physiological effect; that is, the smell acts as a cue that a physiological response is ensuing. Stressor means that a sensation elicits an emotional response in terms of nuisance and thereby provokes a physical stress reaction. For stressors, the context of sensations plays an important role, whereas for markers it is only of minor importance. Taken together, the lack of objective indicators and the role of sensations as stressors pose a problem in the study of environmental hazards: subjective complaints may point toward objective effects or express subjective stress, that is, it remains unclear whether a symptom belongs to the somatic or the psychological spectrum. It is therefore necessary to assess three kinds of cognitions besides objective criteria for somatic complaints[16]: (i) perceived bodily functions/symptoms (ii) perceptions regarding the context of the exposure (eg, sensations) (iii) already processed evaluations (eg, annoying or not), whereby perceived bodily functions also include increased bodily responsiveness and changes in the central nervous system reflected, for example, in changes of cognitive performance. On the other hand, the objective assessment of the exposure is important. As an example, Gabrio et al[18] illustrated the difficulty to find a clear-cut relationship between mold exposure and impaired health due to the entanglement of exposure, smell, nuisance, exposure context, and person-related characteristics.

Practical implications

- The study investigated acute psychological and cognitive effects of exposure to high or low emitting laser printers and included participants who might be regarded as particularly susceptible, both physiologically and psychologically. No evidence for a differential impact of the exposure condition could be found but cognitive processing of the situation and psychological susceptibility seems to play a crucial role. To prevent distress and minimize suffering of the affected persons, we suggest using measures to reduce or eliminate unnecessary exposure.

Against this background, it is no surprise that effects of photocopier or laser printer emissions have been studied with focus either on physiological effects[11,12] or—often under headings like multiple chemical sensitivity (MCS) or idiopathic environmental intolerance (IEI)—on psychological or psychophysiological mechanisms independent of specific hazards.[19-23] Exceptions are two pilot studies that investigated physiological and psychological effects simultaneously: Mersch-Sundermann et al[24] used a real office setting, Luszpinski[25] a controlled exposure with healthy subjects. The office study revealed an increase in respiratory subjective complaints after printing without measureable physiological effects. The controlled exposure study showed no differential effects in physiological measures and a decrease in subjective complaints for both, high and low emitting laser printers, probably due to a better climate in the exposure room compared to the autumn/winter climate in the environment.

Despite these findings, it remains unclear to which extent somatic responses and complaints rest on physiological vs psychological mechanisms or how these mechanisms interact. For example, Eis et al[19] concluded from a multi-center study on MCS that although behavioral accentuations, psychological changes, and psychosomatic problems were dominant over a toxicological-somatic basis, there were still higher somatic complaints found only for actually exposed persons. On the other hand, while Herr et al[26] reported more noticeable changes in physiological parameters for patients with environment-related problems, they could also show that higher somatic complaints were only found for persons who were actually exposed to noxious agents. Regarding cognitive performance, Bornschein et al[27,28] observed no differences between IEI/MCS patients and healthy controls. Finally, Dalton[29] and Dalton and Jaén[30] showed experimentally that even in healthy persons complaints and performance losses could be induced by priming certain odors as dangerous. Thus, cognitive processing seems to be a central mechanism. Despite this, exposure assessment remains important in order not to falsely attribute impairments merely to psychological stressor effects.

The previously reported findings from our exposure study[15] did not provide hints on clinically relevant physiological or acute biochemical differential effects of exposures to either very high or very low levels of ultrafine particles emitted by laser printers. In this study, we

also assessed psychological and performance-related responses which were not included there due to their additional complexity. To separate psychological or mixed psychological/physiological from purely physiological effects, for the present analysis, the sample was categorized in two ways: (i) in symptom-related groups in whom psychological mechanisms due to differences in background and perception of the situation should play a dominant role (ii) in physiologically defined groups in whom effects should be primarily based on physiological mechanisms.

2 | MATERIALS AND METHODS

2.1 | Design and procedure

Study protocol, details of the physiological and biochemical assessments, and their respective results have been described previously.[15] We used a randomized, single-blinded, cross-over experimental design with two exposure conditions (high- and low-level exposure, HLE/LLE, see below) and three primary groups of participants (healthy controls, participants with mild asthma, and participants with self-reported symptoms associated with laser printer exposure [SRS]). Moreover, participants were categorized regarding bronchial hyper-responsiveness (BHR) via methacholine provocation testing. Participants were blinded to the exposure condition and acoustical and visual impressions in the exposure room were comparable for both exposures. Each participant was measured three times on different days: a pretest without exposure and HLE and LLE sessions in randomized order. The pretest was used to assess baseline characteristics including self-reported multiple chemical sensitivity (sMCS) and affectivity (see Section 2.4) and to familiarize participants with the cognitive tests in order to reduce the impact of learning effects. Questionnaires and cognitive performance testing (see Section 2.4) were used to determine psychological effects. The sequence of measurements is shown in Figure 1: Questionnaires (as well as physiological and biochemical assessments not reported here[15]) were administered outside the exposure room in the adjoining examination room. Participants were then seated in the exposure room and took the pretest of cognitive performance. Upon completion, the respective exposure protocol (see below) started. At the end, participants once again completed the cognitive performance

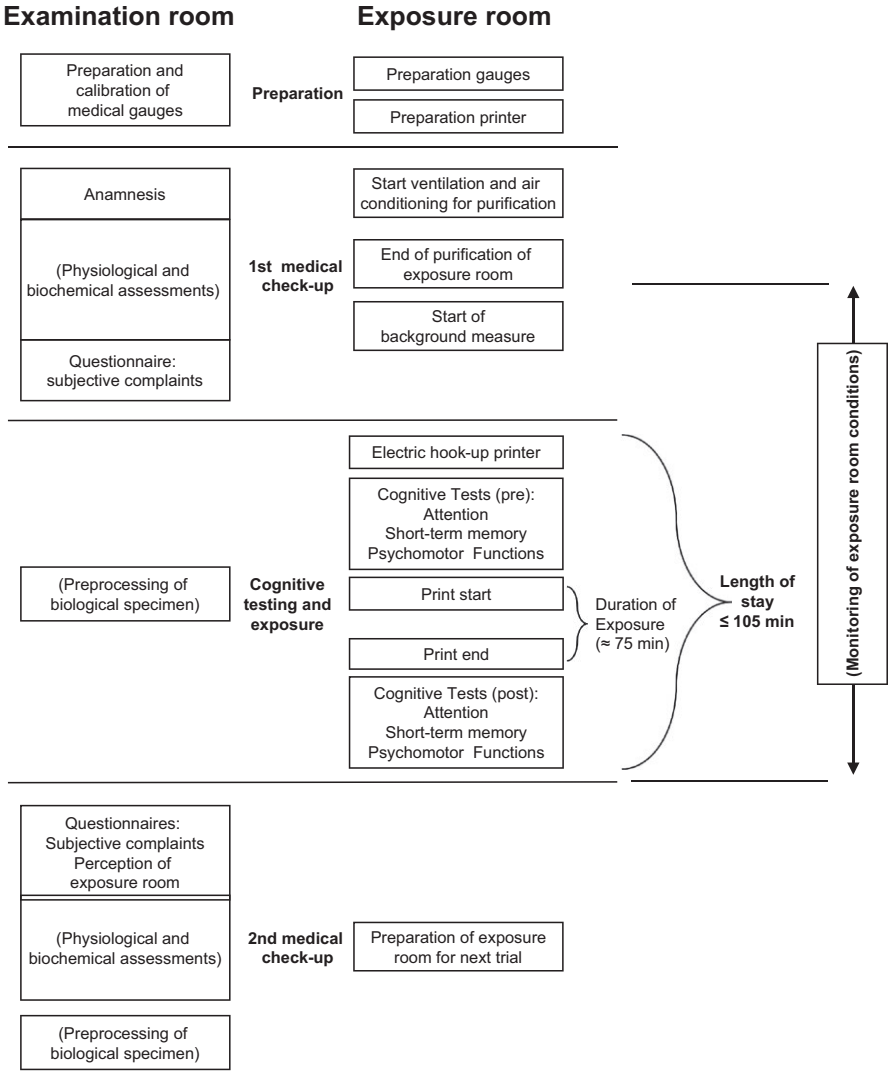

FIGURE 1 Protocol of assessments before, during and after exposures. Note that aspects in parentheses are not mentioned in this article but refer to analyses reported in Karrasch et al[15]

testing and then left the room for further examinations and questionnaires. Overall length of stay in the exposure room was about 105 minutes.

The study was approved by the Ethics Committee of the Faculty of Medicine, Munich University, and written informed consent was obtained from all participants.

2.2 | Exposure

A detailed characterization of the exposure conditions including particle characterization and concentration is given in Karrasch et al[15]. In general, four laser printers—two with very high (high-level exposure, HLE) and two with very low (low-level exposure, LLE) emissions of ultrafine particles—were selected for the exposures from a test pool of printers with known emissions. The exposure room (volume 32 m^3) was unventilated during both exposures. The printers were operated alternatingly and discontinuously within a time span of 75 minutes, that is, each printing only a few pages at a time. After an initial rising phase of 15 minutes, this led to a compensation for particle losses and hence a constant particle number concentration in the last 60 minutes of exposure duration. Particle number concentration (PNC) was continuously monitored by two aerosol spectrometers with high time resolution and overlapping particle size range between 5.6 nm and 25 μm (FMPS Fast Scanning Mobility Sizer type 3090; TSI Inc., Aachen, Germany, and OPC Optical Particle Sizer type 1.108; Grimm Aerosol Technik GmbH, Ainring, Germany). With this procedure, the concentrations of ultrafine particles were kept at approximately 100 000 per cm^3 in HLE. In the LLE sessions, the particle emissions of the two low emitting printers did not measurably increase the background level of ultrafine particles in the exposure room, which was typically 2000-4000 per cm^3. Figure 2 shows the typical time sequence of the particle number concentration for HLE and LLE sessions. Embedded in this sequence was the pretest (during measurement of background level: minutes 0 to ≈15) and the post-test of cognitive performance (during falling phase without printing action: minutes 90 to ≈105).

2.3 | Recruitment and participants

Volunteers were recruited via contacts to healthcare centers and medical specialist practices, newspaper article, bulletins, and by word of mouth.[15] Inclusion criteria were as follows: age 18-60, employed, no corticosteroid therapy within the last 21 days, and non-smokers with a good general state of health. Participants were categorized into three primary groups: healthy controls, subjects with stable mild asthma, and subjects with either specific (eg, dyspnea, cough, eye irritation) or nonspecific (eg, fatigue, poor concentration) self-reported symptoms related to laser printer exposure (SRS). Overall, 52 volunteers participated: 23 healthy controls (11 female; mean age 43.6 ± 12.5, range 20-60 years), 14 with stable mild asthma (9 female; mean age 35.6 ± 11.6, range 21-57 years), and 15 with self-reported symptoms (SRS) related to laser printers (12 females; mean age 47.6 ± 6.8, range 33-58 years). Subjects with SRS were significantly older than the other two groups ($P < .001$).

Although usually asymptomatic, subjects with BHR (according to their methacholine response in a standardized test for methacholine challenge[31]) are more prone of showing obstructive and symptomatic responses to other stimuli and might therefore be particularly susceptible to laser printer emissions. Overall, 28 participants (13 female; mean age 40.7 ± 11.6 years) showed BHR (N = 12 (85.7%) from the group with mild asthma; N = 7 (46.7%) from the SRS group; N = 9 (39.1%) from the healthy control group). Of the participants without BHR (mean age 44.7 ± 11.5 years), 13 were females. Neither sex nor age differences between the BHR defined groups were significant.

2.4 | Measures

To detect potential psychological and cognitive effects of laser printer emissions, a number of questionnaires and psychological tests were used. During the pretest affectivity and multiple chemical sensitivity, two person-related characteristics, were assessed by questionnaire, as both might influence the perception of the exposure situation and the number or quality of reported symptoms. *Negative affectivity* is a relatively stable tendency to experience negative mood or emotions, like pessimism or anxiety, and is often found in persons with impaired health. It was assessed via the positive and negative affect scales (PANAS) by Watson et al[32]. These two scales each comprise ten adjectives on different feelings and emotions. Participants had to mark on a five-point Likert scale (1 = very slightly or not at all, 5 = extremely) to what extent they had felt this way during the last twelve months. Reliability for both positive (Cronbach's alpha = 0.820) and negative affect (Cronbach's alpha = 0.822) was good.

Self-reported multiple chemical sensitivity (sMCS) was assessed with a five-point Likert scale (0 = not at all, 4 = very much) developed by Kiesswetter et al[33,34] which contains eight items on adverse bodily reactions to offensive smells in the environment (eg, "I feel dizzy when I perceive the strong odor of varnish or smoke"). Reliability was very good (Cronbach's alpha = 0.931). sMCS was assumed when one of the eight items was marked with the highest level of agreement.[33]

To check the participants' blinding regarding the type of exposure, three visual analog scales (each 0-100 mm, denoted as scores in the remainder of the paper) were developed assessing *participants' perception of the exposure* immediately after leaving the exposure room (one item each on wellbeing, odor intensity, and odor nuisance in the room[21]).

Subjective complaints were assessed before and after each exposure using a list of 16 symptoms that had to be rated regarding their intensity on visual analog scales (0-100).[35] Symptoms comprised general (eg, dizziness; 4 items, Cronbach's alpha = 0.725), respiratory (eg, dry cough; 9 items, Cronbach's alpha = 0.646), skin (eg, itchiness; 2 items, Cronbach's alpha = 0.792) and eye symptoms (1 item, conjunctival irritation).

To measure *cognitive effects* of exposures, a computer-based, autonomously running test battery (ie, without necessity of instruction or intervention from the experimenter) was developed. It contained established performance tests for cognitive functions that in general might be affected by a relatively short exposure; namely attention and

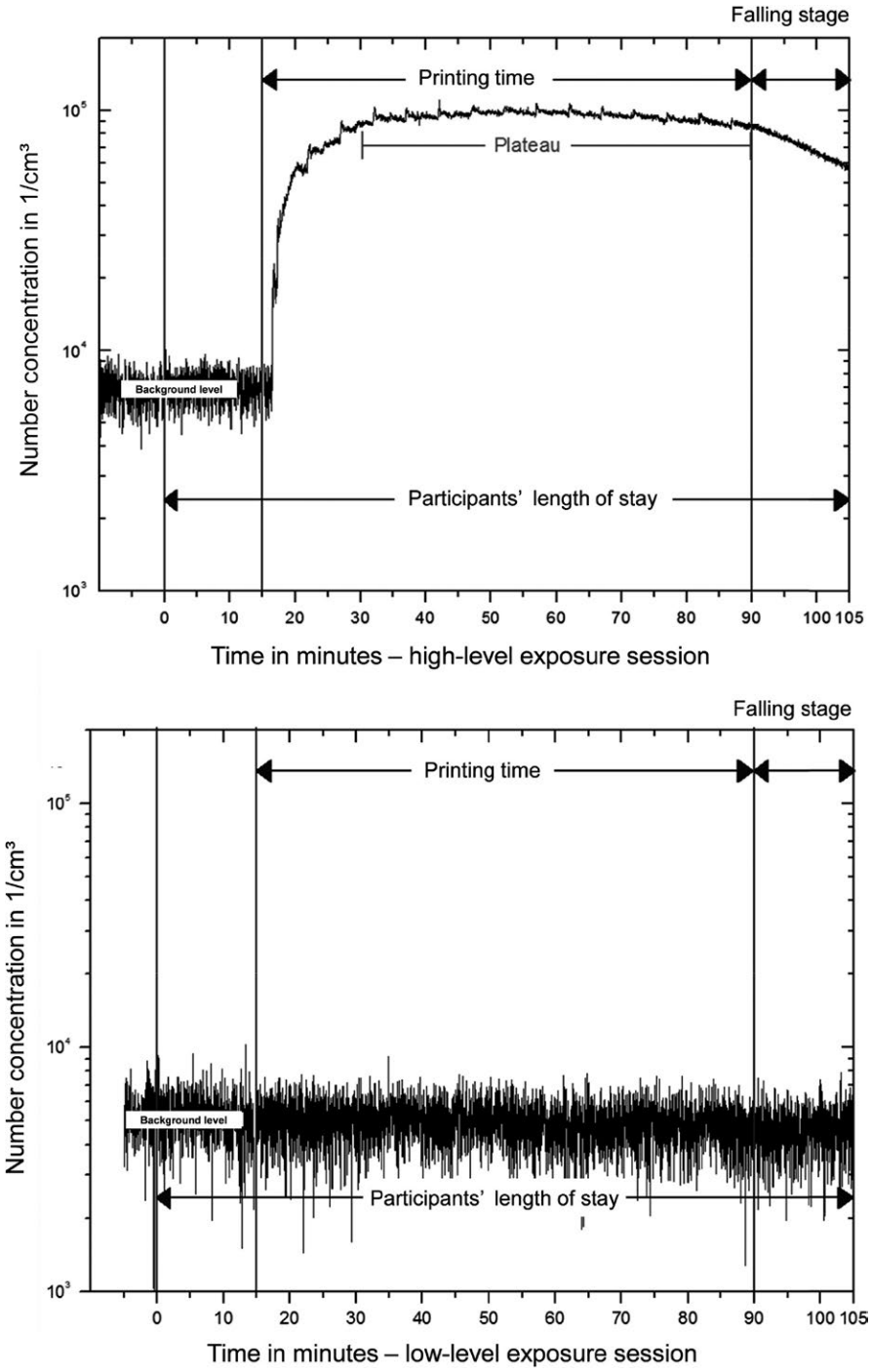

FIGURE 2 Time sequence of typical total particle number concentrations during a high-level exposure session and a low-level exposure session (see also Karrasch et al[15])

concentration, short-term memory, and psychomotor performance. The testing took place in the exposure room during every session immediately before and at the end of exposure (see Figure 1). To minimize learning effects, parallel versions were used in random order for each participant. Test results were directly recorded by the system. The following tests were used and adapted for the study.

Attention and concentration performance was measured with the d2 test of attention.[36,37] The test has good psychometric properties, can be used for a wide age range, and has been applied for a host of medical and psychological research questions.[38-40] It takes about six minutes to complete. Potential effects on short-term memory were assessed using the Benton Visual Retention Test.[41,42] Similar to the d2, the test has good psychometric properties, can be used for a wide age range, and has been applied in a variety of studies.[43-45] It takes about four to five minutes to complete in form M (examinee views a visual design for 10 seconds and then chooses the correct design from a multiple choice of four displays). To assess psychomotor performance, specifically fine-motor hand-eye-coordination skills, a labyrinth test was programmed. In this established group of assessment tests[46] that is also used in medical research on cognition[47,48] persons have to find the optimal way of moving a pen or mouse through a predefined labyrinth as fast as possible and without touching the walls. The test

provides four parameters: solution (cognitive component) and the psychomotor components speed (time to completion), fluency (stopping during passing the labyrinth), and precision (jolting labyrinth walls). In order to strengthen reliability, participants had to perform two labyrinth tests each before and after exposure requiring approximately six minutes. Overall, one trial of the complete test battery took about 17 minutes to complete.

2.5 | Statistical analyses

The difficult recruitment of participants with SRS resulted in a rather small sample size and compromised to some extent the distribution assumptions for parametric testing. Thus, differences between participant groups for variables with single measurements were evaluated using nonparametric tests for independent samples (Mann-Whitney *U* test for comparison of two groups and Kruskal-Wallis test for more than two samples). Likewise, effects for repeated-measurements (exposure effects) were evaluated by the nonparametric Wilcoxon matched-pairs signed-ranks test, firstly, for each emitter condition separately by paired comparisons of the pre-and post-exposure data, and secondly, by comparing the pre-post differences between emitter conditions.

Additionally, to test for interaction effects, three-way repeated-measures analyses of variance (ANOVA) were used whereby the repeated factor was measurement time (pre-post exposure) and the between-factors were participant group (either the three primary groups or the two BHR groups) and exposure condition (high-level exposure vs low-level exposure). Main effects, two-way and three-way interactions are reported and compared with additional nonparametric comparisons of simple main effects, that is, comparison of each factor level while keeping the other factors constant. This allows for an additional evaluation whether detected interactions really exist in the sample.

Most fluid cognitive abilities are known to decline with age when tested—as in our study—with context-free tests.[49] As age differed between groups (participants with SRS being significantly older), we adjusted for age in all cognitive test results and report unstandardized residuals. To integrate the parameters of the labyrinth tests, a composite measure was developed that combines solution, speed, fluency, and precision in the following way: number of solved labyrinths (0-2) + (1-((number of stops + number of jolts)/speed) * 10), whereby higher values indicate better performance and the psychomotor component is valued higher than the purely cognitive component. Time was limited to 180 seconds maximum.

All analyses were performed with SPSS 23. The level of statistical significance was set at $P = .05$. We did not include corrections for multiple testing but provide exact p-values as far as possible.

3 | RESULTS

3.1 | Sample characteristics

The three primary study groups differed systematically regarding self-reported multiple chemical sensitivity (sMCS) and affectivity (Table 1). Healthy participants reported significantly lower sMCS as well as higher positive and lower negative affect than both, participants with mild asthma and participants with SRS. Moreover, using the criterion for a diagnosis of sMCS proposed by Kiesswetter,[33] a difference between groups can be seen. While in the healthy subjects only one person (4.3%) fulfilled this criterion, this was true for four subjects with mild asthma (28.6%) and for eight participants with SRS (53.3%) ($P = .002$). Regarding the secondary categorization of participants into groups with and without BHR, neither sMCS nor affectivity showed significant differences between groups.

3.2 | Perception of exposure situation

Before analyzing differential effects of the group on the perception of the situation in the cross-over design, we first checked the blinding of the participants by comparing the perceived odor intensity and nuisance at the first exposure session: Participants who experienced the HLE in the first session reported on a scale from 0 to 100 about the same intensity (mean 41.0 ± 34.4) and nuisance (mean 24.3 ± 29.2) as participants who experienced the LLE in the first session (odor intensity mean 39.7 ± 33.0; odor nuisance mean 23.6 ± 27.2; $P = .85/.87$, respectively).

As the blinding was effective, we tested for main effects of the exposure condition and the primary participant group as well as their interaction using a two-way ANOVA. For none of the exposure perception variables (ie, wellbeing in the exposure room, odor intensity,

TABLE 1 Differences in self-reported multiple chemical sensitivity (sMCS), and positive and negative affect between the primary participant groups

		Healthy controls, N = 23 (a)		Mild asthma, N = 14 (b)		Subjects with SRS, N ≤ 15 (c)		Overall difference between groups*
	Range	Mean	Standard deviation	Mean	Standard deviation	Mean	Standard deviation	*P* values
Positive affect	1-5	3.08[b,c]	0.83	2.12[a]	0.84	2.49[a]	0.78	.005**
Negative affect	1-5	2.06[b,c]	0.93	3.21[a]	0.75	2.93[a]	0.81	.001***
sMCS	0-32	4.17[b,c]	3.59	8.79[a]	6.27	15.00[a]	9.88	.001***

*Kruskal-Wallis tests for comparisons of all three participant groups; **error probability 0.1–1%, ***error probability ≤ 0.1%; [a-c]significant differences (Mann-Whitney *U* tests, $P \leq .05$) between the respective groups.

odor nuisance), a significant main effect of the exposure condition or an interaction effect was found. However, there were strong main effects for the participant group (wellbeing $P < .001$; odor intensity $P = .001$; odor nuisance $P < .001$).

Non-parametric comparisons (see Table 2) showed that most differences were triggered by the participants with SRS in the HLE condition who reported the lowest well-being of all groups and the highest odor nuisance. For odor intensity, this also held true, with the exception of subjects with mild asthma in the LLE condition. In the HLE condition, subjects with SRS did not differ from the LLE condition in which they also perceived the situation differently from the healthy controls in the LLE and HLE condition. Taken together, subjects with a history of symptoms related to laser printer exposure upon inclusion showed a stronger response to the exposure situation than healthy subjects or subjects with mild asthma.

For the secondary categorization of participants into the two groups with and without BHR, no differences in the perception of the exposure situation were found.

3.3 | Subjective complaints

The intensity of *subjective complaints* in terms of general, respiratory, skin, and eye-related symptoms was assessed before and after each exposure with visual analog scales (ranging from 0 to 100). To show the general symptom load, sum values were computed. With a potential maximum of 1600, overall symptom load before (mean 48.7 ± 64.7; range 0-315) and after all exposures (mean 60.1 ± 72.8; range 0-366) was low. In detail, respiratory symptoms (maximum 900) had a mean sum value 33.3 ± 46.0 before and of 32.6 ± 51.3 after exposures. General symptoms (maximum 400) reached a mean sum value of 7.9 ± 17.6 before and of 16.6 ± 26.3 after exposures. The skin symptoms (maximum 200) had a mean sum value of 3.5 ± 8.2 before and of 4.2 ± 8.0 after exposures. Eye-related symptoms (maximum 100) summed up to 4.0 ± 7.8 before and 8.7 ± 14 after exposures (descriptive values for individual symptoms can be found in Table S1). Therefore, the overall level of reported symptoms was low but rather variable.

To compare all symptoms on a common denominator, the following analyses use mean values for the different subjective complaints. The full three-way repeated-measures ANOVA showed significant main effects of the primary group (all $P \leq .007$) for all areas of symptoms. With the exception of respiratory ($P = .991$) and skin symptoms ($P = .277$), main effects of measurement time (pre vs post) were also significant (all $P \leq .002$). Moreover, significant interaction effects between the pre-post-test and groups were found for all symptom domains and overall symptoms, except for skin (all $P < .001$; general symptoms $P = .010$), in such a way that in most instances subjects with SRS reported increases in both exposure conditions, whereas the other groups did not or to a smaller degree. No main effects for exposure condition or other interactions were found. Table 3 shows the results of the non-parametric testing of changes in reported symptoms.

Overall, changes were very small and, as with the analysis of variance results, no significant differential effect between LLE and HLE

TABLE 2 Comparison of interaction effects of participant group and low-level (LLE) and high-level exposure (HLE) on the perception of exposure

	Healthy/LLE, N = 23 (a)		Healthy/HLE, N = 23 (b)		Mild asthma/LLE, N = 14 (c)		Mild asthma/HLE, N = 14 (d)		Subjects with SRS/LLE, N = 14 (e)		Subjects with SRS/HLE, N = 15 (f)		Overall difference between groups*
(Range per aspect 0-100)	Mean	Standard deviation	Mean	Standard deviation	Mean	Standard deviation	Mean	Standard deviation	Mean	Standard deviation	Mean	Standard deviation	*P* values
Well-being	$75.5^{e,f}$	24.8	$82.1^{e,f}$	12.6	76.0^{f}	21.6	71.1^{f}	19.0	$60.9^{a,b}$	24.4	$55.1^{a,b,c,d}$	22.7	.003**
Odor intensity	$20.3^{c,e,f}$	22.5	26.7^{f}	30.5	38.4^{a}	28.2	34.6^{f}	26.0	39.6^{a}	31.9	$60.3^{a,b,d}$	30.7	.002**
Odor nuisance	$6.1^{c,d,e,f}$	8.5	$11.1^{d,e,f}$	16.5	$16.9^{a,f}$	17.2	21.8^{f}	20.4	$35.5^{a,b}$	31.3	$47.3^{a,b,c,d}$	34.6	<.001***

*Kruskal-Wallis tests for comparisons of all six groups; **error probability 0.1–1%, ***error probability ≤ 0.1%; [a-f]significant differences (Mann-Whitney *U* tests, $P \leq .05$) between the respective groups.

TABLE 3 Changes in reported symptoms after low-level exposure (LLE) and after high-level exposure (HLE)

	LLE			HLE			Difference between LLE and HLE changes[a]
Symptoms (maximum value per domain 100)	Mean change after LLE	Standard deviation of change	Pre vs post[a] *P* values	Mean change after HLE	Standard deviation of change	Pre vs post[a] *P* values	*P* values
Respiratory symptoms							
All	−0.5	5.90	.228	0.4	3.92	.971	.088
Healthy controls	−0.6	1.80	.406	−0.0	1.52	.544	.062
Subjects with mild asthma	**−3.6**	**6.14**	**.009****	**−1.8**	**3.03**	**.034***	.542
Subjects with SRS	2.8	8.29	.326	3.2	5.49	.121	.636
General symptoms							
All	**1.6**	**3.40**	**.000*****	**3.0**	**5.38**	**.000*****	.089
Healthy controls	**0.5**	**1.24**	**.024***	**1.2**	**2.50**	**.022***	.125
Subjects with mild asthma	**2.0**	**3.86**	**.010***	**3.8**	**6.74**	**.004****	.970
Subjects with SRS	**3.0**	**4.74**	**.003****	**4.9**	**6.60**	**.000*****	.143
Skin symptoms							
All	0.0	2.13	.885	0.6	3.00	.275	.216
Healthy controls	0.0	0.58	.867	0.7	3.81	.886	.663
Subjects with mild asthma	−0.8	3.31	.438	−0.0	2.08	1.000	.250
Subjects with SRS	0.9	2.15	.180	0.8	2.35	.219	.640
Eye-related symptoms							
All	**3.8**	**11.94**	**.016***	**5.5**	**15.69**	**.020***	.893
Healthy controls	2.8	7.68	.059	1.5	6.56	.498	.204
Subjects with mild asthma	−3.1	9.05	.266	3.2	11.41	.910	.241
Subjects with SRS	**12.3**	**15.37**	**.004****	**13.7**	**24.56**	**.013***	.946
Overall symptoms							
All	0.4	3.66	.739	**1.4**	**3.36**	**.004****	.053
Healthy controls	0.0	1.35	.930	0.5	1.51	.113	.170
Subjects with mild asthma	**−1.8**	**2.77**	**.015***	0.1	2.86	.960	.060
Subjects with SRS	**3.2**	**5.18**	**.025***	**4.0**	**4.44**	**.001*****	1.00

[a]Wilcoxon test; *error probability 1–5%, **error probability 0.1–1%, ***error probability ≤ 0.1%; *P* values <.05 are highlighted in bold text.

could be observed, although there was a tendency across all groups regarding overall symptoms ($P = .053$). Eye-related symptoms increased in all subjects in both exposure conditions; however, this effect was mainly triggered by the subjects with SRS. Similarly, an increase in overall symptoms mainly for the HLE conditions was found in the complete sample, again, mainly driven by the SRS group. Subjects with mild asthma reported a significant decrease in respiratory symptoms during both exposures. General symptoms increased in all groups and during both exposures, whereby the relatively largest increase occurred for subjects with SRS. Figure 3 presents mean changes of overall symptoms between pre- and post-test for all groups and exposure conditions and visualizes the additional effect that subjects with mild asthma reported a decrease in overall symptoms in the LLE condition (Table 3).

For the secondary categorization of participants with and without BHR, an interaction effect in respiratory symptoms was found in such a way that independent of exposure condition participants without BHR reported an increase in symptoms (pre-post difference 1.3), whereas participants with BHR reported a decrease (pre-post difference −1.1, $P < .05$). Moreover, for participants without BHR, there was a significant increase in eye-related symptoms in LLE (pre-post difference 7.3) but not in HLE (pre-post difference 6.1), although the difference in the pre-post-change of these symptoms was not significant ($P = .39$), and no effect was found for participants with BHR. General symptoms increased in both groups and independent of exposure condition (pre-post differences between 1.1 and 3.8; all $P < .05$). Taken together, there were hardly any systematic differences regarding the secondary categorization of participants according to BHR and the exposure condition.

3.4 | Cognitive performance

For the d2-test of attention/concentration[36,37] and the Benton test of short-term memory,[41,42] there are normal values derived from the

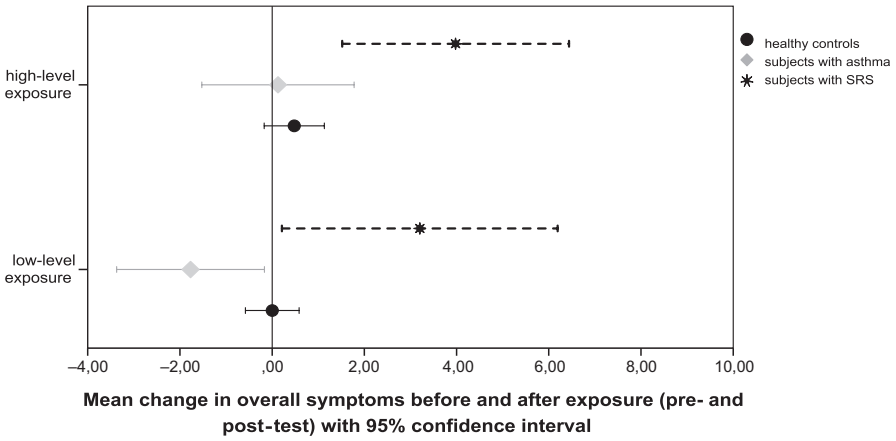

FIGURE 3 Change of overall symptoms between pre- and post-test in high-level and low-level exposure conditions for primary participant groups. Note that mean change could vary between −100 and +100 but varies in fact only between −1.8 and +4.0 (therefore, scaling of *x*-axis was reduced)

general population. To describe the general level of cognitive performance in the different groups, age-adjusted percentile ranks at pre-test were calculated (see also descriptive absolute values and residuals for cognitive performance in Table S2). For attention performance, healthy controls reached percentile ranks between 2 and 97 with a median value of 75 (mean 69 ± 24.7), subjects with mild asthma ranked between 3 and 100 with a median value of 83 (mean 69 ± 31.4), and subjects with SRS showed percentile ranks between 4 and 95 with a median value of 52 (mean 58 ± 23.5). For short-term memory performance, all groups reached percentile ranks between 40 and 90 and median values of 90, only means and standard deviations varied (mean for healthy controls: 82 ± 12.9; for subjects with asthma: 81 ± 15.2; for subjects with SRS: 74 ± 18.2). Thereby, upon inclusion, no differences in short-term memory performance between groups at the beginning of the trial were found but lower percentile ranks in attention performance in subjects with SRS. It has to be noted that the percentile ranks are representative of the general population in the respective age range, and thus, even the lowest median rank of 52 was completely on average, as 52 percent of the population perform worse than participants with SRS did. More obvious are the very high percentile ranks in attention performance in the other two groups—this has to be taken into account when interpreting results.

Results of the three-way repeated-measures ANOVA showed no main or interaction effects of primary group, exposure condition, and measurement time on attention performance (all $P > .20$). For short-term memory performance, a significant three-way interaction ($P = .007$) and an accompanying two-way interaction between group and exposure condition ($P = .048$) was found. These effects were mainly driven by increases in the HLE condition in the healthy controls and subjects with mild asthma, whereas performance of subjects with SRS decreased in this condition. For psychomotor performance, no interaction effects and a main effect of the group occurred ($P = .004$) in such a way that subjects with SRS performed worse than healthy controls before and after each exposure. Results of the non-parametric test of changes in Table 4 mirror these results, that is, differential changes for HLE and LLE were only found in the group of participants with SRS in such a way that non-significant increases in LLE were contrasted with a significant decrease (psychomotor performance) and a tendency for decrease (memory performance) in HLE (see Figure 4). However, the effects for memory and psychomotor performance mainly depended on the spreading of performance at pre-testing (see Table S2).

For the secondary categorization of participants with and without BHR, the full ANOVA model showed only one significant effect for psychomotor performance—a two-way interaction between measurement time (pre-post) and BHR group ($P = .044$) indicating that independent of exposure condition participants without BHR performed the labyrinth tests better after exposure, whereas subjects with BHR performed worse. Nonparametric tests of changes showed no significant differences whatsoever within each exposure condition or between LLE and HLE.

4 | DISCUSSION

In this study, the biochemical and physiological changes after exposure to either very low or very high laser printer particle emissions were absent or low.[15] They did not indicate a coherent pattern of changes in the three participant groups or suggest clinically relevant differential effects between high-level and low-level exposure. The present article complements these previous findings by addressing psychological and cognitive changes. It concentrates on the psychological characteristics of the participants, their perception of the exposure situation as well as on changes in subjective complaints and cognitive performance. For the psychological characteristics of affectivity and sMCS, a consistent pattern emerged: Healthy controls showed the lowest negative affect, highest positive affect, and lowest sMCS scoring. They diverged systematically from participants with mild asthma or SRS. These two groups—as to be expected by the strain imposed by health impairments—did not differ regarding affectivity, but more than half of the subjects with SRS would be "labeled" with sMCS and only about one-fourth of those with mild asthma. The additional categorization according to BHR was performed to define groups with and without possible physiologically based susceptibility to particle effects without any discernible (respiratory) symptoms. Results on the psychological characteristics are in line with this, as there were no significant differences between the groups.

TABLE 4 Changes in cognitive performance after low-level exposure (LLE) and after high-level exposure (HLE)

	LLE			HLE			Difference between LLE and HLE changes[a]
Age-adjusted unstandardized residuals	Mean change after LLE	Standard deviation of change	Pre vs post[a] *P* values	Mean change after HLE	Standard deviation of change	Pre vs post[a] *P* values	*P* values
Attention performance (d2)							
All	−1.46	31.55	.389	1.44	31.50	.708	.162
Healthy controls	3.30	37.89	.988	5.65	30.15	.119	.380
Subjects with mild asthma	−5.42	20.75	.426	−2.63	35.24	.670	.715
Subjects with SRS	−5.33	29.94	.502	−1.23	31.30	.720	.303
Memory performance (Benton)							
All	0.01	1.12	.664	−0.01	1.26	.552	.964
Healthy controls	**−0.32**	**0.82**	**.044***	0.16	1.08	.917	.097
Subjects with mild asthma	−0.07	0.81	1.00	0.29	1.30	.358	.539
Subjects with SRS	0.64	1.56	.261	−0.56	1.39	.081	**.021***
Psychomotor performance (labyrinth tests)							
All	0.04	0.57	.477	−0.04	0.48	.512	.359
Healthy controls	0.08	0.52	.445	0.08	0.50	.687	.988
Subjects with mild asthma	−0.13	0.37	.268	−0.02	0.53	.952	.426
Subjects with SRS	0.14	0.79	.268	**−0.23**	**0.36**	**.041***	**.049***

[a]Wilcoxon test; *error probability 1-5%; *P* values <.05 are highlighted in bold text.

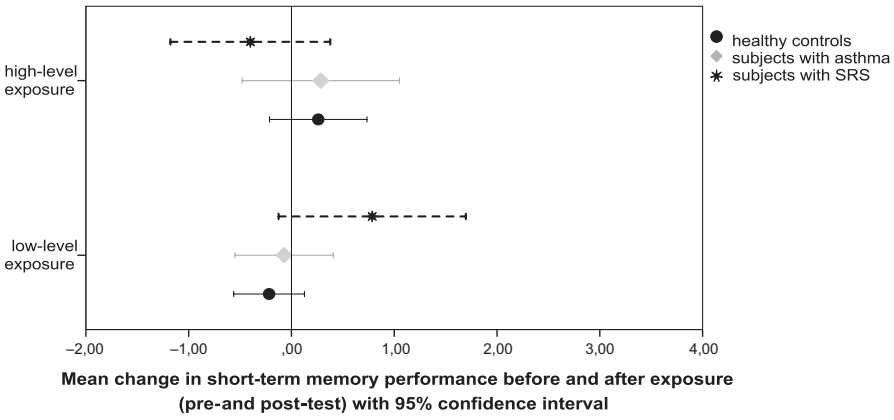

FIGURE 4 Change of short-term memory performance between pre- and post-test in high-level and low-level exposure conditions for primary participant groups. Note that mean change could vary between −15 (strongest possible decrease in memory performance) and +15 (strongest possible increase) but varies in fact only between −0.4 and +0.8 (therefore, scaling of *x*-axis was reduced)

The results on the perception of the exposure situation were also dependent on the participants groups: In general, subjects with SRS showed the strongest responses to the exposure situation. Independent of exposure condition (LLE/HLE), this group reported the lowest well-being and the highest odor intensity and nuisance, whereby odor intensity was perceived slightly stronger for HLE than for LLE. These effects are psychologically plausible, as subjects with SRS had to surrender to a situation for which they assumed a detrimental pathogenic effect. This assumption is confirmed by the secondary categorization according to BHR which showed no difference between groups.

For subjective complaints, we used visual analog rating scales that were applied before and after exposures and allowed for a detailed assessment. Overall, the level of symptoms in different domains was so low that a clinical relevance can be excluded. There was an overall trend toward a higher increase in symptoms during HLE compared to LLE. However, this trend was mainly caused by a proliferation of eye-related symptoms in subjects with SRS. Besides this observation, there are no results that point toward a differential impact of the exposure condition on subjective complaints. In general, the highest increase in complaints was found in subjects with SRS irrespective of exposure. This suggests that the mere fact of being exposed to laser printer

emissions was sufficient to increase symptoms even though the low emitting laser printers produced no quantifiable contribution to the background level of fine and ultrafine particles in the exposure room. Contrary to this clear result pattern, there were hardly any systematic differences between participants with and without BHR. Aside from the study design, we found the relatively highest changes for eye irritation. On the one hand, this is the symptom most often mentioned in the clinical history of subjects with SRS in association with printer exposure. On the other hand, a causal relationship of this symptom with the climatic conditions in the exposure room as well as with the computer work during the cognitive tests, that is, independent of laser printer emissions, can be assumed. An analogous reasoning is applicable to general symptoms (ie, dizziness, headache, circulatory disturbances, nausea), for which the relatively second highest changes were found across all groups and exposures.

In contrast to the differences between groups regarding their baseline characteristics, their perception of the exposure situations, and their subjective complaints, there were only few and weak differences regarding cognitive performance. Potential three-way interactions between group, exposure condition, and pre-post measurement indicate that only for the group of participants with SRS, an increase in short-term memory and psychomotor performance in the LLE condition occurred, in contrast to a decrease in the HLE condition. However, these effects rest upon the rather high variation of performance observed already at pre-test so that an unambiguous interpretation is not possible. Regarding the secondary categorization according to BHR again no systematic effects were found, in particular no differential effects of the exposure condition.

An experimental study such as the present one necessarily has limitations regarding the duration and number of exposures as well as the observation period after exposures. Our study comprised a single, although very high, short-term exposure and covered effects occurring within 2 hours after exposure. Although this limits conclusions on potential chronic effects of laser printer emissions, as both adaption and cumulative effects seem possible, the symptoms typically reported after exposure by subjects with SRS are of acute nature. That is, it should have been possible to detect such changes by our design and methods. Moreover, the sample size of this study was quite high compared to conventional exposure studies, although small regarding the potential variability between subjects which also may limit the generalizability of the results. Despite this, the study had maximum power owing to its cross-over controlled design. Moreover, it is unique in including participants with self-reported symptoms due to laser printer emissions as well as subjects with mild asthma.

Taken together, we found no consistent evidence for a differential short-term influence of high and low emitting laser printers on psychological and cognitive aspects. In fact, the majority of results—notably those referring to psychological processing of situations such as perception, causal attribution, and evaluation[30,50,51]—could be traced back to subjects with SRS who reacted differently to the exposure situation than the other participants. This is in line with previous studies on MCS or on patients with environment-related problems[19,26] which could not identify a clear toxicological-somatic basis. Instead, the combination of both exposure and its perception as harmful, noxious, or toxic[29,30] are likely to play an important role in the development of symptoms, that is, cognitive processing seems to be a central mechanism. We aimed to separate physiological causes and psychological processing using two different categorizations of participants: On the one hand, by including subjects with reported symptoms due to laser printer emissions and, thus, perceivable suffering, and on the other hand, a purely physiologically defined group based on bronchial hyperresponsiveness in which potential effects should be caused exclusively by physiological mechanisms. As the latter showed no systematic differences whatsoever with regard to the exposure conditions, psychological processing seems to be the most relevant factor in the response to laser printer emissions. Following the transactional stress model by Lazarus and Folkman,[52] it could be assumed that based on different personal backgrounds of participants with SRS, the entire exposure situation is experienced as a threat leading to a number of physiological and psychological stress reactions. In contrast, healthy subjects or subjects with mild asthma perceive the situation as irrelevant or challenging. In addition to trait anxiety and focus of attention as relevant aspects of cognitive processing in IEI/MCS patients,[53] this type of stress-related explanation suggests to investigate the learning history of patients with SRS more closely in order to understand what types of situations or trigger events lead to a negative primary and secondary appraisal and in the long run to an enhanced psychological susceptibility.

In view of these considerations and despite the fact that clinically relevant biochemical and physiological acute effects of high-level exposure to laser printer emissions were not observed in this study,[15] it seems reasonable to use measures to reduce or eliminate unneeded exposures in order to prevent distress and lessen the burden of suffering for affected persons.

5 | CONCLUSION

The present randomized controlled study aimed to separate physiological and psychological effects of laser printer emissions. Therefore, persons who reported symptoms from exposure to laser printer emissions and participants with mild asthma were included as groups that might be especially susceptible to laser printer emissions, and additionally, a purely physiologically based categorization of susceptibility to particle effects (bronchial hyperresponsiveness) was used. With the exception of strong differences in participant characteristics, all other effects regarding subjective complaints and cognitive performance were either very small or inconsistent and could mostly be traced to participants with self-reported symptoms associated with laser printer emissions. Neither physiological susceptibility nor exposure level showed differential acute effects whatsoever. Although the study design does not allow conclusions regarding potential effects of long-term exposure, the findings suggests in line with our results on physiological effects[15] that further research on laser printer emissions should focus more strongly on psychological vulnerability and its preconditions for developing laser printer emission-related symptoms.

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SUPPORTING INFORMATION

Additional Supporting Information may be found online in the supporting information tab for this article.

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