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Electromagnetic Hypersensitivity: Evidence for a Novel Neurological Syndrome

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ABSTRACT

Objective: We sought direct evidence that acute exposure to environmental-strength electromagnetic fields (EMFs) could induce somatic reactions (EMF hypersensitivity). *Methods*: The subject, a female physician self-diagnosed with EMF hypersensitivity, was exposed to an average (over the head) 60-Hz electric field of 300 V/m (comparable with typical environmental-strength EMFs) during controlled provocation and behavioral studies. *Results*: In a double-blinded EMF provocation procedure specifically designed to minimize unintentional sensory cues, the subject developed temporal pain, headache, muscle twitching, and skipped heartbeats within 100 s after initiation of EMF exposure ($p < .05$). The symptoms were caused primarily by field transitions (off–on, on–off) rather than the presence of the field, as assessed by comparing the frequency and severity of the effects of pulsed and continuous fields in relation to sham exposure. The subject had no conscious perception of the field as judged by her inability to report its presence more often than in the sham control. *Discussion*: The subject demonstrated statistically reliable somatic reactions in response to exposure to subliminal EMFs under conditions that reasonably excluded a causative role for psychological processes. *Conclusion*: EMF hypersensitivity can occur as a *bona fide* environmentally inducible neurological syndrome.

KEYWORDS: electromagnetic fields, evoked potentials, hypersensitivity, provocation study, sensory transduction, sleep study

INTRODUCTION

Man-made electromagnetic fields (EMFs) such as those produced by cell phones, powerlines, or computers are ubiquitous in the general and workplace environments. About 3%–5% of the population subjectively associates acute or subacute exposure to EMFs with departures from normal function or feeling (EMF hypersensitivity) (Levallois, Neutra, Lee, & Hristova, 2002; Schreier, Huss, & Röösli, 2006). The prevalence of self-reported EMF hypersensitivity has usually been attributed to somatization disorders (Rubin, Das Munshi, & Wessely, 2005; Rubin, Nieto-Hernandez, & Wessely, 2010).

A possible nonpsychological basis for EMF hypersensitivity was provided by the discovery of the ability of human beings to detect weak EMFs, as evidenced by the occurrence of field-onset and field-offset brain potentials (Carrubba, Frilot, Chesson, & Marino, 2007), and the induction of steady-state changes in brain electrical activity that persisted during the presence of the field (Marino, Carrubba, Frilot, Chesson, & Gonzalez-Toledo, 2010). The underlying mechanism of field sensory transduction appears to be an electric-force-sensitive ion channel (Marino, Carrubba, Frilot, & Chesson, 2009). Animal studies suggest that the electroreceptor cells and/or afferent processing cells are located in the brain stem (Frilot, Carrubba, & Marino, 2009, 2011).

Despite the physiological and biophysical evidence that could explain at least some cases of human somatic responses to EMFs without invoking psychological processes (Carrubba et al., 2007; Frilot et al., 2009, 2011; Marino et al., 2009, 2010), direct evidence of nonpsychological EMF hypersensitivity is lacking. Our purpose was to determine whether EMFs could

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produce symptomatic responses in a putatively hypersensitive subject while appropriately controlling for chance, confounders, and somatization.

METHODS

Subject

In the context of ongoing human, animal, and biophysical studies involving EMF sensory transduction in our laboratory, we were contacted by a 35-year-old female physician with multiple neurologic and somatic symptoms including headaches, hearing and visual disturbances, subjective sleep disturbances and nonrestorative sleep, and musculoskeletal complaints, all of which she reported could be precipitated by exposure to environmental EMFs and abated by withdrawal from the fields. Among the environmental triggering sources she identified were cell phones, computers, powerlines, and various common electrical devices. During extensive interviews she credibly explained the reasons for her belief that EMFs from common environmental sources could provoke her symptoms.

After she agreed to medical tests appropriate for evaluating her medical condition, she was admitted as a patient on the neurology service and underwent a physical exam including a comprehensive neurologic exam, a clinical electroencephalogram (EEG) exam, a noncontrast magnetic resonance (MR) imaging of the brain, an overnight sleep study (with video and expanded EEG montage) in which the resulting polysomnogram was scored in accordance with standardized rules (American Academy of Sleep Medicine, 2007), a standard laboratory evaluation of serum electrolytes and blood chemistries, liver function tests, serum fasting cortisol, and complete blood count, and direct evaluations of her EMF sensitivity in a series of EMF provocation and behavioral studies (see below). The institutional review board at the LSU Health Sciences Center approved all experimental procedures, and the subject gave her written informed consent.

EMF Exposure

The subject sat in a comfortable wooden chair with her eyes closed, and uniaxial 60-Hz (unless noted otherwise) sinusoidal electric fields were generated by applying a voltage to parallel 49-cm square metal plates spaced 36 cm apart (Figure 1). The equipment that controlled the field was located outside the subject's view and emitted no visual or auditory stimuli. The background electric field (the field present irrespective of whether or not a voltage was applied to the parallel plates) was about 1 V/m throughout the region occupied by the subject (HI-

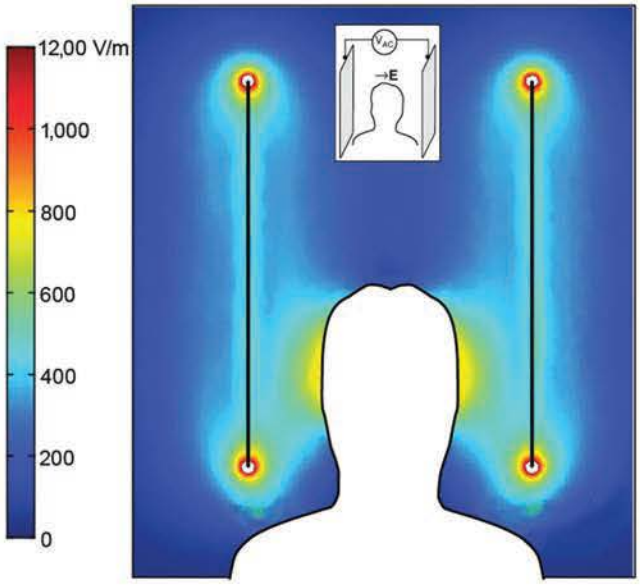

FIGURE 1. Spatial distribution of the external electric field (E) in the mid-sagittal plane. E was generated by applying $V_{AC} = 100$ volts to parallel metal plates while the subject was electrically isolated (insert), and calculated at all points in the subject's environment. Average E surrounding the head was about 300 V/m.

3603, Holaday, Eden Prairie, MN, USA). The plate arrangement did not produce magnetic fields. The continuously present background 60-Hz magnetic field was 0.1 mG, and the geomagnetic field was 599.8 mG, 68.4° below the horizontal component (component along the direction of the applied field, 360.5 mG) (MAG-03, Bartington, GMW, Redwood City, CA, USA). High-frequency signals from cell-phone towers and other distant antennae (1–10 GHz) were less than 0.1 μW/cm^2 (the background fields in the sleep-study room were similar; (Spectran, Aaronia, Euscheid, Germany).

In the provocation studies the electric field was applied for 100-s intervals with a duty cycle of 50% and a repetition rate of 10 Hz, which resulted in alternating field-on and field-off pulses of 100 ms (pulsed field); a continuous field (100% duty cycle) was used in one of the provocation studies. Duty cycle, pulse structure, and interval length were regulated by a microcontroller programmed to produce the desired signals. When the duty cycle was 50%, the actual EMF stimuli consisted of (1) 10 onset stimuli per second $\times$ 100 s = 1,000 field-onset stimuli per interval; (2) an equal number of field-offset stimuli; and (3) the presence of the EMF for a total of 50 s. When the duty cycle was 100%, there was only one field-onset stimulus and one field-offset stimulus, and the EMF was present for 100 s. In the behavioral studies, the electric field was applied in trials consisting of a 2-s epoch when a pulsed field was applied (50%

duty cycle, 10-Hz repetition rate) and a 10-s field-free control epoch.

Field Strength

The applied electric field was significantly distorted by the subject's body, resulting in strong inhomogeneities in the field surrounding the subject. To overcome the problem of measuring the external field, we used Maxwell's laws to calculate it at every point in the subject's vicinity. The subject was modeled as an electrically isolated composite of rectangular solids representing the trunk and lower extremities and an ellipsoid representing the head. The assumed conductivity was 1 S/m. The total electric field at every point was determined for $V_{AC} = 100$ V using finite-element analysis consisting of approximately 10^6 elements; a more detailed mesh was automatically generated in the head region (Multiphysics, Comsol, Los Angeles, CA, USA). The peak external electric field was about 1,000 V/m (see Figure 1); the average field was about 300 V/m around the head and less than 50 V/m around the body. The peak and average field strength and duration of exposure were far below the levels generally recognized as capable of producing physiological effects in human subjects (International Commission on Non-Ionizing Radiation Protection, 1998).

The external electric field resulted in an induced internal electric field in the brain in accordance with physical law. The strength of the induced brain electric field was comparable with that induced by environmental-strength power-frequency electric and magnetic fields (Carrubba, Frilot, Chesson, & Marino, 2010; Carrubba, Frilot, Hart, Chesson, & Marino, 2009).

Somatic Responses

A pulsed field (50% duty cycle) was applied for 100 s in 10 independent field-exposure intervals. The controls were ten 100-s sham-exposure intervals during which a field was not applied. The order of the field and sham intervals was determined randomly. The environmental conditions during the field-exposure and sham-exposure intervals were identical except that the wires carrying the plate voltage were disconnected during the sham-exposure intervals. At the end of each interval the subject was questioned by an interviewer blinded to whether or not the field had been applied and asked to describe any symptoms she developed during the interval, whether or not the symptoms had persisted into the interview period. She was queried using descriptive terms she had employed. Whenever she reported symptoms, commencement of the next interval was delayed until she reported that they had abated.

We used a pulsed field because we expected it would result in a stronger symptomatic response compared with a continuous field (Carrubba, Frilot, Chesson, & Marino, 2008; Frilot et al., 2011). To test this reasoning, we performed a second study to assess whether the subject developed a differential symptomatic response to the pulsed and continuous fields. The subject was exposed or sham exposed for 100-s intervals and immediately after each interval was interviewed as described above. A sham (S) field, continuous (C) field (100% duty cycle), and pulsed (P) field (50% duty cycle, 10 Hz) were applied, and the SCP pattern was repeated five times. The subject was blinded regarding the use of different EMFs; from her perspective, the laboratory procedures were identical to those followed in the first study. The interviewer was aware that the effects of C and P fields were being compared but was blinded regarding the actual sequence of the fields.

Behavioral Responses

We considered the possibility that any symptomatic response might be a result of the combined processes of conscious awareness of the EMF followed by a somatization reaction based on a fear that EMFs were harmful. We approached the issue by determining whether the subject could consciously perceive a field when it was presented in multiple independent trials. A field having the same strength and spatial distribution as previously (Figure 1) was applied in a series of trials each of which consisted of a 2-s epoch during which a pulsed field (50% duty cycle, 10-Hz repetition rate) was applied and a 10-s field-free control epoch. Eight independent sequences were employed, each with 30–50 trials. In three sequences, the frequency was 60 Hz; in two, it was 1 kHz; and in three others, the respective frequencies were 10, 100, and 500 kHz.

The subject held a small plastic box that housed a buzzer, a button labeled YES and another button labeled NO. In the middle of each on and off epoch the buzzer emitted a 4-kHz tone at 60 dB that lasted 100 ms, and she was instructed to press the YES or NO button whenever she heard the tone, depending on whether or not she had any conscious sensation of a field at that moment. Employing a custom-designed virtual instrument (LabView, National Instruments, Austin, TX, USA), we determined the number of YES and NO responses in the presence and absence of the field in each sequence. In addition, four sham sequences (minimum of 30 trials in each) were conducted in which a field was not applied. The subject had no knowledge that an off–on pattern was being used in the field sequences or that some sequences consisted of sham exposure.

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TABLE 1. Polysomnography results. Comparison with usual night, per patient: "Same as usual." No epileptiform activity noted during arousals associated with unintended gross motor activity. Normal REM-related atonia

	Subject	Normal range
Sleep latency	6 min	13.4 ± 10.1 (Hirshkowitz, Moore, Hamilton, Rando, & Karacan, 1992)
Stage N1 sleep	13.8%	3%–8% (Chokroverty, Thomas, & Bhatt, 2005)
Stage N2 sleep	51.8%	44%–55% (Chokroverty et al., 2005)
Stage N3 sleep	23.6%	10%–15% (Chokroverty et al., 2005)
Stage R sleep	10.7%	20%–25% (Chokroverty et al., 2005)
REM latency	150.5 min	57%–66 min (Pressman, 2002)
WASO index	6/hr	1.3 ± 0.8 (Hirshkowitz et al., 1992)
WASO total	40.5 min	10.7 ±11 min (Naifeh, Severinghaus, & Kamiya, 1987)
Total sleep time	340.5 min	340.0 ± 70 (Hirshkowitz et al., 1992)
Sleep efficiency	88%	86.4% ± 11.6% (Hirshkowitz et al., 1992)
Arousal index	34.2/hr	16.8 ± 6.2 (Bonnet & Arand, 2007)
PLM index	7.8/hr	< 5/hr (Nicolas, Michaud, Lavigne, & Montplaisir, 1999)
AH index	0.2/hr	< 5/hr (American Academy of Sleep Medicine, 2005)

Note: REM, rapid eye movement; WASO, wake after sleep onset; PLM, periodic limb movement; AH, apnea/hypopnea.

Statistics

The frequencies of the somatic and behavioral responses in the presence and absence of the field were evaluated using the chi-square test (2 × 2 tables) or the Freeman–Halton extension of the Fisher exact probability test (2 × 3 tables; Freeman & Halton, 1951).

RESULTS

Clinical Studies

The patient's physical examination was unremarkable. The presence of frequent subjective awakenings from sleep, sometimes with unintended gross motor activity such as muscle twitching and leg jerking, prompted clinical concern for a sleep-related movement disorder, parasomnia, or nocturnal epilepsy. The polysomnogram revealed significant sleep fragmentation and discontinuity (Table 1) but no evidence of significant sleep-disordered breathing, nocturnal epilepsy, or abnormal rapid-eye-movement-related (REM-related) atonia. Periodic limb movements were noted but did not appear to be a major sleep-disrupting force.

Standard and 24-hr video-accompanied EEG recordings revealed normal-appearing background rhythms and no epileptiform activity. EEG performed in the presence of active cellular telephone use provoked a right-sided headache, but produced no unusual EEG waveforms. The MR image revealed evidence of cortical dysplasia in the right temporal lobe, and right parietal polygyria, both without interval change when compared with a study performed 19 months earlier. Laboratory evaluation for common metabolic/endocrine problems and blood count abnormalities was unremarkable.

Somatic Responses

The sequence and characteristics of the symptomological and behavioral experiments are shown in Table 2.

The question of a relation between the presence of the field and the occurrence of symptoms was directly addressed by interviewing the subject immediately following 100-s field-exposure or sham-exposure intervals; both the interviewer and the subject were blinded regarding the exposure condition. During the interviews, the subject reported a range of symptoms including localized pain in her jaw, ear, or the side of her head, a more diffuse head pain, and muscle pain or twitching in the hip, neck, and back. Sometimes she qualified the symptom as "strong" or "mild," and sometimes she denied all symptoms. We grouped the symptoms related to localized head pain as "temporal pain," those related to diffuse head pain as "headache," and those related to muscle effects as "muscle pain/twitching." Symptoms reported more rarely were indicated explicitly (see Table 3a). The subject consistently reported pronounced symptoms that occurred during the field intervals, particularly in intervals 7, 13, 14, 15, and 18. In the sham intervals, she reported no symptoms in intervals 4, 6, 8, 16, and 20; weak temporal pain in intervals 2, 3, and

TABLE 2. Sequence and characteristics of experiments

Experiment	Electric field Condition	No. of trials	Trial Duration (sec)	Response
1	Pulsed	10	100	Symptoms
	Sham	10	100	
2	Pulsed	5	100	Symptoms
	Continuous	5	100	
	Sham	5	100	
3	Pulsed	300	1	Behavior
	Sham	150	1	

TABLE 3. Evaluation of the relation between presentation of a pulsed electric field and the development of symptoms. (a) Results from the individual 100-s exposure intervals. (b) Summary table

(a) Interval no.	Condition	Result
1	Pulsed field	Temporal pain
2	Sham	Mild temporal pain
3	Sham	Mild temporal pain
4	Sham	No symptoms
5	Pulsed field	Temporal pain; headache
6	Sham	No symptoms
7	Pulsed field	Skipped heartbeats; feeling unease
8	Sham	No symptoms
9	Pulsed field	Headache
10	Sham	Mild headache
11	Pulsed field	Temporal pain
12	Sham	Mild headache
13	Pulsed field	Muscle twitch; feeling unease
14	Pulsed field	Strong headache
15	Pulsed field	Strong headache
16	Sham	No symptoms
17	Pulsed field	Stiff neck
18	Pulsed field	Muscle twitch; temporal pain
19	Sham	Mild temporal pain
20	Sham	No symptoms

(b) Field condition	Symptoms: None	Mild	≥ Mild
Sham	5	5	0
Pulsed field*	0	0	10

*$p < .05$.

19; and a weak headache in intervals 10 and 12. The field and sham distributions of symptoms differed significantly ($p < .05$; see Table 3b).

In a second study, the relative role of EMF changes (number of onsets and offsets) and steady-state presence of the EMF were directly addressed by interviewing the subject immediately following 100-s exposure intervals in which either a pulsed field or a continuous field was presented. She was queried regarding her symptoms as previously and reported symptoms in both field intervals (see Table 4a). The symptoms triggered by the pulsed field were more intense compared with the sham control ($p < .05$; see Table 4b); the symptoms triggered by the continuous field did not differ from the sham control ($p = .16$). The subject reported no symptoms in four of five sham intervals (intervals 1, 4, 10, 13).

TABLE 4. Evaluation of the comparative effect of continuous and pulsed fields relative to a sham field on the development of symptoms. (a) Results from individual 100-s exposure intervals. (b) Summary table

(a) Interval no.	Condition	Result
1	Sham	No symptoms
2	Continuous field	No symptoms
3	Pulsed field	Temporal pain
4	Sham	No symptoms
5	Continuous field	No symptoms
6	Pulsed field	Mild headache
7	Sham	Mild headache
8	Continuous field	Muscle twitch
9	Pulsed field	Severe pain
10	Sham	No symptoms
11	Continuous field	Temporal pain
12	Pulsed field	Headache; muscle twitch
13	Sham	No symptoms
14	Continuous field	Mild temporal pain
15	Pulsed field	Mild temporal pain

(b) Condition	Symptoms: None	Mild	≥Mild
Sham	4	1	0
Continuous field	2	0	3
*Pulsed field	0	2	3

*$p < .05$.

Behavioral Responses

The possible influence of conscious awareness of the EMF on the development of symptoms was investigated by assessing whether the subject could consciously perceive the field. A total of 300 independent trials involving carrier frequencies of 60 Hz to 500 kHz were used; the controls consisted of 150 sham trials. The results did not depend on the carrier frequency, and consequently the data were combined for analysis (see Table 5).

The subject failed to respond to the tone seven times while the field was on and seven times while it was off, resulting in a total of 293 responses for each of the two conditions. There were no missed responses in the sham trials. The overall YESresponse rate in the field trials was $(51/586) \times 100 = 8.7\%$. The occurrence of a YES response was significantly associated with the presence of the field ($p < .05$; see Table 5a), but the sensitivity of the YES responses was low ($[32/(32 + 261)] \times 100 = 11\%$). The YES response rate in the sham trials was slightly higher than that seen in the field trials ($[27/273 = 9.9\%]$) (see Table 5b).

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Table 5. Evaluation of conscious perception of a pulsed electric field. The subject's responses during the presence (on) and absence (off) of the field, respectively

		Pulsed field	
(a)	Response	On	Off
	Yes*	32	19
	No	261	274
		Sham	
(b)	Response	On	Off
	Yes	15	12
	No	135	138

*$p < .05$.

DISCUSSION

Appropriately controlled provocation studies are required to establish the existence of EMF hypersensitivity and to understand the relative importance of psychological and nonpsychological processes in mediating any observed symptoms. A working laboratory definition of EMF hypersensitivity formulated in symptomological terms is therefore needed to permit recognition of hypersensitivity reactions when they occur. In previous provocation studies, the assumption was made that true hypersensitive subjects would exhibit more or less the same symptoms in response to repeated provocations. The assumption led to experimental designs that involved averaging across exposed and control groups, which is an inherently insensitive statistical procedure for detecting real but variable responses (Rubin et al., 2005, 2010). The assumption is particularly inapplicable to EMF hypersensitivity because intrasubject and intersubject variabilities are its salient features (Levallois et al., 2002; Schreier et al., 2006). We defined EMF hypersensitivity as the occurrence of any medically recognized symptom in response to provocation using an environmentally relevant EMF; there was no requirement that the same symptom must reoccur when the EMF provocation was repeated. This definition avoided the problem of masking real effects and more appropriately matched the laboratory procedure to the known characteristics of EMF hypersensitivity (Levallois et al., 2002; Schreier et al., 2006). We focused on a single self-reported subject and employed a procedure in which she served as her own control. While controlling for artifacts, chance, and somatization, the question whether she reliably exhibited *any* symptomatic responses to an EMF was addressed; the alternative hypothesis was that she did not exhibit EMF-triggered symptoms. The laboratory conditions were controlled in such a way that a putative role of psychological processes could reasonably be identified.

The subject developed symptoms in association with the presentation of a pulsed electric field significantly ($p < .05$) more often than could reasonably be explained on the basis of chance (see Table 3). Several considerations suggested that the statistical link was a true causal association with a subliminal EMF. First, the subject's environment was carefully controlled to avoid putative confounding factors. The testing took place in an acoustically quiet environment, and the presence of uncontrolled environmental EMFs was nil. The environmental conditions during the field-exposure and sham-exposure intervals were identical except that during the sham-exposure intervals, at a point far removed from the subject's field of view, the wires carrying the plate voltage were disconnected. A key aspect of our laboratory procedure was the elimination of sensory cues that could serve as conscious markers of the electric field leading to a somatization reaction. All appropriate precautions were taken to eliminate potential confounders. Second, the occurrence of symptoms was significantly associated with the type of EMF (see Table 4). The symptomatic response was associated with the pulsed EMF, which maximized occurrence of the number of transient changes in the EMF (off–on and on–off), not with the presence of the field, as expected on the basis of prior animal studies where the issue of somatization was irrelevant (Frilot et al., 2011). Finally, in a behavioral study specifically designed to assess awareness of the field, YES response rates were 8.7% and 9.9% in the field and sham conditions, respectively, which provided no evidence for a psychological role in the development of the subject's symptoms. We therefore conclude with a reasonable level of certainty that the causal association we found between the presence of the EMF and the subject's symptoms was mediated by a subconscious neural process. Although chance was an unlikely explanation for the association, that possibility could not be excluded. The existence of the neurological syndrome reported here was previously suspected but not documented.

The mechanism for the subject's symptoms of headache, visual disturbances, and somatic musculoskeletal discomfort following exposure to EMFs is unknown. On the basis of clinical evaluation, intermittent seizure activity is not a credible explanation, although a deeper epileptic focus with partial seizure activity may have escaped the detection of surface EEG electrodes. The abnormal findings in the subject's medical workup included the abnormal MR image (cortical dysplasia and polygyric changes) and extensive sleep discontinuity and fragmentation manifested in the overnight polysomnogram; the possible association of these

findings with the subject's syndrome of EMF hypersensitivity is unknown.

Our aim here was to concentrate on the previously unaddressed question whether acute exposure to weak EMF could produce real but not precisely predictable somatic effects mediated by nonpsychological processes. Within the limitations of the study, we concluded that we demonstrated the neurological syndrome in the subject we studied. The question of whether EMF hypersensitivity is a significant public-health problem was not addressed here. The EMF we employed was equivalent in strength and pulse structure to EMFs pervasively present in the environment (Levallois et al., 2002; Schreier et al., 2006), and our results were consistent with the possibility that environmental EMFs can directly trigger clinical symptoms. Nevertheless resolution of the public-health issue depends on a deeper understanding of how internal EMFs caused by environmental EMFs are related to physiological process and of the role of psychological factors and comorbidities in the exposed population in exacerbating the processes resulting in disease.

Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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