Effects of Compact Fluorescent and Incandescent Lights on Brain Electrical Activity

P. Arefjev
A. Chalkovskaja
R. Mameniškienė
Clinics of Neurology and Neurosurgery, Faculty of Medicine, Vilnius University; Center of Neurology, Vilnius University Hospital Santariskiu Clinics, Lithuania

Summary. Background. During widespread use of compact fluorescent lamps (CFL), noticeable number of patients and some international societies have claimed that CFL have or could have negative influences on psychoneurological disorders. European Scientific Committee on Emerging and Newly Identified Health Risks in 2008 stated that more research is needed to establish whether CFL constitute any higher risk than incandescent lamps. The lack of evidence on whether CFL may trigger disturbances of brain activity prompt this investigation.

Objective. To compare effects of compact fluorescent and incandescent lights on brain electrical activity and clinical manifestation.

Methods. It was experimental single blind study, performed in Vilnius University Hospital Santariskiu Klinikos between November 2009 and January 2010. 35 individuals aged 20–30 years were included according to defined criteria. All subjects were randomly assigned to one of the two lighting groups: compact fluorescent or incandescent. In all individuals standard video-electroencephalogram (EEG), modified with reading and short/long term exposure to light, was recorded. EEG analysis was based on comparison of alpha wave (occipital derivations) mean frequency (F) and peak amplitude (A) variation differences between two groups. Additionally, intergroup prevalence of EEG alterations and subjective, objective signs during and after light exposure, were detected.

Results. 25 EEG records were used for final analysis. The CFL illuminated group consisted of 14 persons, 3 of them had history of central nervous system (CNS) disorder. The incandescent light group consisted of 11 persons, 1 of them had CNS disorder. The two study groups did not differ significantly by age, gender, duration of last sleep, non-eating time, total words read and the number of errors answering text comprehension questions (p > 0.45). Higher A and F was detected after the long-term exposure to CFL (p <0.05). The prevalence of EEG alterations (paroxysmal theta activity, sporadic theta and sharp waves) was higher in CFL group (50.0% vs. 9.1%, p = 0.042). Subjective and objective symptoms did not differ significantly between two groups (p ≥ 0.313).

Conclusions. Long-term exposure to CFL in comparison to incandescent light has different effects on brain electrical activity. Estimated higher prevalence of EEG alterations, distinct alpha frequency and peak amplitude in CFL group suggest irritant effects of CFL on the brain.

Keywords: compact fluorescent lamps, energy saving bulbs, incandescent lamps, video-electroencephalogram, alpha waves.

Neurologijos seminarai 2011; 15(47): 18–25

INTRODUCTION

Since the end of the 20th century, when compact fluorescent lamps (CFL) increasingly gained production popularity among lamp manufacturers becoming dominant source of lighting in household, significant number of patients and some international societies claimed that CFL have or could have negative effects on psychoneurological disorders [1–3]. The most frequently described conditions
Effects of Compact Fluorescent and Incandescent Lights on Brain Electrical Activity

which could be aggravated under the influence of CFL are epilepsy, migraine, myalgic encephalomyelitis, multiple sclerosis, dyspraxia, autism spectrum disorders, fibromyalgia, electro sensitivity, and traumatic brain injury [1–5, 7, 9]. However, there are considerably more unspecified and still not recognized symptoms triggered by this light, such as headache, chronic fatigue, sleep/memory problems, depressive/aggressive tendencies, etc. [4, 6, 8].

Due to increasing number of complaints arising from CFL users, European Scientific Committee on Emerging and Newly Identified Health Risks (SCENIHR) in 2008 established a scientific rationale on whether CFL constitute any higher risk than incandescent lamps (IL). The committee tried to determine which lamp characteristics are responsible for the health risks: light spectrum specificities, UV radiation, flicker, electromagnetic fields. Unfortunately, no suitable direct evidence on the relationship between CFL and any of mentioned before health conditions has been identified. Of all CFL properties, only UV radiation was determined as a potential risk factor for the aggravation of the light-sensitive dermatological disorders. The committee states that there is still insufficient scientific evidence to make the final conclusions on CFL [10].

The present experimental study was undertaken to compare effects of compact fluorescent and incandescent lights on brain electrical activity and clinical manifestations in order to determine possible differences and prompt further scientific investigations.

METHODS

It was experimental single-blind study performed in Vilnius University Hospital Santariškių Klinikos between December 2009 and January 2010.

Subjects

The study was carried out on 35 healthy adult volunteers without neurologic complaints. They were selected by mother language (Lithuanian), age (20–30 years), clinical eye refraction (normal or ≥ -3.5 D, when vision correction lenses are unnecessary for reading in normal distance (30–40 cm)). All subjects were free of any medication. All participants were assigned to one of the two lighting groups (CFL or IL) using block randomization with “AABB” sequence. Prior to the experiment, the subjects were informed about experimental procedure explaining that the real aim of the study remains unrevealed until the end of the study, so persons could not affect results. Each participant signed an informed consent form.

Light source

Two types of illumination sources were provided to the subjects during experiment. We selected 60 Watt (W) standard shape frosted incandescent light bulb with light output of 710 lumen (lm). Compact fluorescent light bulb we used had a power rating of 11W, was spiral-shaped with light output of 670 lm and color temperature of 2700 Kelvin (warm white light) which is similar to light color and intensity produced by conventional 60W lamp.

EEG technical features

Electroencephalograms were recorded in an electromagnetically shielded room by a Nihon Kohden Neurofax EEG-1100 EEG machine, with lower and upper band-pass filter limits set at 0.5Hz and 30Hz, respectively. Ag/AgCl electrodes with impedance less than 5 kΩ were placed over the subject’s scalp according to the International 10–20 system. Overall, 26 electrodes with 2 electrodes recording eye blinking and 2 electrodes for I standard ECG derivation were used.

EEG procedure

In all individuals standard video-electroencephalogram modified with reading and short/long term exposure to light was recorded after 3:00 p.m. Duration of each EEG recording was 1 hour 10 minutes. During the examination all windows were darkened, all artificial light sources and mobile phones were switched off. The only light source could be the display of working computer.

Control EEG signal was recorded when persons were lying with closed (4 min) and opened (4 min) eyes. Then short-term (1 min) exposure to particular light was applied during lying with closed eyes. In the sequel long-term (reading for 30 min) exposure to light was applied. During reading tested persons were seated, raising the head of the bed at an angle of 70 degrees. The light source was located in the subject’s left side, 70–80 cm from the text. Depending on the group, the text was illuminated randomly with compact fluorescent or incandescent light. Reading text was of neutral content, the same for all subjects, typed in 2 columns using Times New Roman font, 12 pt size, 1.15 line spacing in order to minimize eyes’ movement and reduce influence on subjective symptoms. After reading subjects were returned into initial lying position. EEG activity was recorded continuously with closed (4 min) and opened (4 min) eyes, then provocative hyperventilation (HV) (3 min) and intermittent photic stimulation (IPS) (3 min) tests were applied. IPS was performed at rates of 1, 5, 10, 15, 20, 25, 30, 50, 60, 33, 23, 13 Hz (8 s for each frequency: 4 s with closed and 4 s with opened eyes), the time interval between each session was 8 s with opened eyes. Finally, 10–15 min was given to sleep (figure 1). After EEG record each person answered fifteen text comprehension questions and filled specially prepared questionnaire.

Data analysis

For EEG evaluation we selected alpha waves, oscillations in the frequency range of 8 to 13 Hz, prominent in right and left occipital derivations when the subject’s eyes are
closed. EEG analysis was based on comparison of alpha wave mean frequency (F) and peak amplitude (A) variation differences between two groups, estimated intergroup prevalence of EEG alterations, assessment of subjective and objective (blink rate, heart rate) signs during and after light exposure.

Control alpha pattern frequency (F1, in Hz) and peak amplitude (A1, in mV) were measured during the first 4 minutes of EEG data section when person was lying with closed eyes. The successive F and A measurements have been done at these EEG data sections: during short-term (1 min) exposure to light (F2, A2); after long-term exposure to light (30 min) on the first, second and third minute of EEG signal, averaging F and A on this data section (F3, A3); during 3 min period after hyperventilation (F4, A4) and intermittent photic stimulation (F5, A5). Subsequently all measured parameters were compared with control F1 and A1, computing difference values (F1 – Fn, A1 – An, where „n“ is a certain EEG data section). Obtained values were compared between CFL and IL groups, using independent-samples T-test. All measurements have been done using artifact-free EEG data sections applying ‘Zoom’ window with ‘Analyze’ function.

All EEG recordings were independently assessed by an experienced epilepsy specialist for revealing abnormal changes. Blink induced artifacts in EEG data sections with opened eyes were calculated creating blink rate parameter. Blink rate during long-term exposure to light at the 2nd, 29th minute was compared with control EEG data section. Heart rate was also counted on the control and exposed to light EEG data sections according to number of QRS complexes in ECG. Obtained values were compared likewise. Computed difference values then were compared between two lighting groups.

Table 1. Baseline characteristics

<table>
<thead>
<tr>
<th>Baseline variable</th>
<th>Mean ± SD or %</th>
<th>Comparison of groups, p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>IL group (n=11)</td>
<td>CFL group (n=14)</td>
</tr>
<tr>
<td>Age, years</td>
<td>22.73 ± 1.62</td>
<td>22.57 ± 2.5</td>
</tr>
<tr>
<td>Male sex</td>
<td>18.2% (n=2)</td>
<td>21.4% (n=3)</td>
</tr>
<tr>
<td>CNS disorder in history</td>
<td>9.1% (n=1)</td>
<td>21.4% (n=3)</td>
</tr>
<tr>
<td>Duration of last sleep, h</td>
<td>7.36 ± 1.75</td>
<td>6.75 ± 2.14</td>
</tr>
<tr>
<td>Non-eating time, h</td>
<td>8.45 ± 4.06</td>
<td>8.68 ± 5.11</td>
</tr>
<tr>
<td>Total words read</td>
<td>4692.7 ± 739.5</td>
<td>4930.9 ± 2355.7</td>
</tr>
<tr>
<td>The number of errors answering text comprehension questions</td>
<td>1.64 ± 1.36</td>
<td>1.93 ± 2.13</td>
</tr>
<tr>
<td>Control blink rate, blink per min</td>
<td>17 ± 13.25</td>
<td>11.93 ± 9.0</td>
</tr>
<tr>
<td>Control heart rate, beat per min</td>
<td>66.9 ± 8.4</td>
<td>73.21 ± 7.56</td>
</tr>
<tr>
<td>Control alpha wave frequency in right occipital derivation, Hz</td>
<td>11.5 ± 1.43</td>
<td>10.86 ± 1.29</td>
</tr>
<tr>
<td>Control alpha wave frequency in left occipital derivation, Hz</td>
<td>11.96 ± 2.2</td>
<td>10.86 ± 1.1</td>
</tr>
<tr>
<td>Control alpha wave peak amplitude in right occipital derivation, mV</td>
<td>76.84 ± 26.62</td>
<td>82.46 ± 26.84</td>
</tr>
<tr>
<td>Control alpha wave peak amplitude in left occipital derivation, mV</td>
<td>63.52 ± 27.28</td>
<td>75.44 ± 25.47</td>
</tr>
</tbody>
</table>

CFL – compact fluorescent light, IL – incandescent light.
Statistical data analysis was performed using SPSS 16.0 software package. The Kolmogorov-Smirnov test was used to test variables for normal distribution. Differences in demographic, clinical, EEG parameters between two lighting groups were evaluated by Fisher’s exact test (for categorical variables) and Student’s T-test (for continuous normally distributed variables). Values of $p<0.05$ were considered statistically significant.

**RESULTS**

According to defined criteria 25 EEG records were used for final analysis. 10 records were excluded due to technical interference and artifacts. There were 5 male and 20 female in our study group. Age ranged between 20 and 30 years with a mean of 22.6 years. For other baseline characteristics see table 1.

The CFL group consisted of 14 persons, 3 of them had history of central nervous system (CNS) disorder. The IL group consisted of 11 persons, 1 of them had CNS disorder. Subjects with history of traumatic subdural haemorrhage, brain contusion, autism spectrum disorder, bacterial meningitis were considered as hypersensitive due to reported possible increased sensitivity to fluorescent light [2–5, 9]. Although no abnormalities were revealed in control EEG of these persons.

The two study groups did not differ significantly by age, gender, duration of last sleep, non-eating time, total words read, the number of errors answering text comprehension questions ($p \geq 0.45$). Difference in control EEG parameters (control blink rate, heart rate, alpha wave frequency and peak amplitude in right and left occipital derivations) was also not significant between two groups ($p \geq 0.06$).

We estimated tendency of alpha wave maximal amplitude increase in right occipital derivation during short-term (1 min) exposure to CFL: the mean difference of peak amplitude (A1-A2 (dex)) between control and impacted alpha waves was $16.85 \pm 18.55$ in IL group and $-3.125 \pm 23.91$ in CFL group, $p=0.1$ (table 2).

Higher maximal amplitude and frequency were detected after the long-term exposure to CFL during reading. Significant difference of peak amplitude was estimated in right occipital derivation (A1-A3 ( dex)), 7.2 vs. -8.0 in IL and CFL group respectively; $p=0.005$. Frequency differed significantly in left occipital derivation (F1-F3 (sin), 0.82 vs. -0.16 in IL and CFL group respectively; $p=0.05$).

![Figure 2. Samples of EEG recordings: presumable differences of amplitude and frequency in alpha band due to exposure to different light](image)
### Table 3. Comparison of changes in blink rate and heart rate between two groups during long-term exposure to light

<table>
<thead>
<tr>
<th>Difference of parameters</th>
<th>Mean difference values ± SD</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>IL group (n=11)</td>
<td>CFL group (n=14)</td>
</tr>
<tr>
<td>Blink rate: control - 2</td>
<td>3.45 ± 10.86</td>
<td>5.57 ± 7.27</td>
</tr>
<tr>
<td>Blink rate: 2 – 29</td>
<td>0.27 ± 5.76</td>
<td>-1.79 ± 3.49</td>
</tr>
<tr>
<td>Blink rate: control - 29</td>
<td>3.73 ± 12.61</td>
<td>3.79 ± 8.51</td>
</tr>
<tr>
<td>Heart rate: control - 2</td>
<td>-4.73 ± 5.53</td>
<td>-3.14 ± 6.94</td>
</tr>
<tr>
<td>Heart rate: 2 – 29</td>
<td>-2.18 ± 1.94</td>
<td>-2.07 ± 5.08</td>
</tr>
<tr>
<td>Heart rate: control - 29</td>
<td>-6.91 ± 4.81</td>
<td>-5.21 ± 7.76</td>
</tr>
</tbody>
</table>

Control – measurement at control data section, 2 – measurement during 2nd minute of long-term exposure to light, 29 – measurement during 29th minute of long-term exposure to light. **CFL** – compact fluorescent light, **IL** – incandescent light.

**Figure 3. Subjective symptoms during long-term exposure to particular light**

CFL – compact fluorescent light, IL – incandescent light.

There was also noticeable tendency of maximal amplitude increase in CFL group in the left occipital derivation (p=0.067). Although there were no significant differences between two groups after hyperventilation (p>0.17), significant peak amplitude difference appeared again after intermittent photic stimulation in the right occipital derivation (A1-A5 (dex), 15.1 vs. 2.7 in IL and CFL group respectively; p=0.035) (Table 2). Summarizing obtained results we want to notice alpha wave frequency acceleration and amplitude increase in persons exposed to CFL (figure 2).

The prevalence of nonspecific EEG alterations was higher in CFL group (1 vs. 7 in IL and CFL group respectively, p=0.042). Only 1 person (without history of CNS disorder) from IL group was identified as having sporadic theta waves in right parietooccipital area (O2-P8 derivations) after long-term exposure to IL, whereas there were 7 participants in CFL group in whom certain EEG alterations occurred. One participant with history of brain concussion had theta wave series in occipital derivations (O2>O1) during long-term exposure to CFL (reading).

Sporadic waves of different localization were detected in 3 participants (1 – P4, P8, O2; 2 – F7, T7; 3 – T7 derivations) mostly after long-term exposure to CFL, also after IPS. Paroxysmal generalized theta activity lasting for 0.5–1 s appeared and repeated several times with growing frequency from 0.5 to 5 events per 10 minutes in 3 subjects during and after long-term exposure to CFL. One of these persons had traumatic subdural haemorrhage in history.

According to subjective symptoms experienced during long-term exposure to light participants were divided into two groups: those who haven’t noticed any symptoms (n=15), and those with one or more noted symptoms (n=10). No significant difference was found between two lighting groups (p=0.414). Several symptoms were described both in IL and CFL group. However, general fatigue (n=4) and itchy eyes (n=3) were mentioned only after exposure to CFL (figure 3).

We also haven’t found significant differences of blink rate and heart rate variation on the 2nd and 29–30th minute of reading under illumination between two lighting groups (p>0.313) (table 3).

**DISCUSSION**

Despite of growing dissatisfaction among CFL customers during the past decade, only few studies reported on neurological health risks related to fluorescent light [11–15]. To be more specific, it is still unclear what investigation methods should be used to provide reliable scientific evidence. The majority of performed studies analyse single technical characteristics of CFL: electromagnetic fields, spectrum, flicker, UV radiation. Obtained results are often extrapolated to all known biological impacts of discrete parameter. However, it is important to understand that all factors act in combination, thus experimental, cohort human studies could give indispensable information.

In our study we tried to determine possible differences in brain electrical activity during exposure to IL or CFL by using conventional EEG test. The idea originated due to expanding list of unspecified troublesome CFL-related neurologic symptoms arising from consumer society. The present investigation demonstrates different lighting effects on the brain as we considered before. Detected tendency of maximal amplitude increase during 1 min exposure to CFL is rather argumentative. It is possible to suppose that such difference might have been developed due to early impact of lighting on the brain electrical activity. However, CFL use electronic ballasts which generate significant levels of electromagnetic fields (EMF). The radiated emissions may cause electronic interference with
other devices, like EEG machine [16]. Therefore, after long-term exposure to light alpha band parameters were measured in the data section when the light was switched off, thus eliminating EMF effects on the device. Nevertheless, significant differences of maximal amplitude and frequency were detected in right and left occipital derivations respectively. This means that CFL surely have different influence on the human brain, compared to IL. However, these findings are difficult to explain. It is known that higher-frequency and lower-amplitude EEG states are associated with increased level of cortical arousal [17]. In our particular case CFL illumination increased both frequency and peak amplitude. Notwithstanding, we are inclined to consider irritant neurological effects of CFL. Moreover, estimated higher prevalence of EEG alterations in CFL group also supports this idea. Detected intermittent slow activity patterns, such as theta wave series, paroxysmal generalized theta wave bursts are nonspecific and can be seen in association with a wide variety of pathological processes [18–19]. These alterations are also described in patients after traumatic brain injury [18, 20]. Nevertheless, clinically appearance of abnormal slow activity in otherwise normal EEG correlates with impairment of attention and arousal [21]. Sharp waves are usually found as random focal discharges. The occurrence of such findings in apparently healthy individuals requires reasonable interpretation. The EEG assessment of large healthy populations showed paroxysmal discharges in 0.3–5.1% of all adult controls [20, 22–25]. Particularly, in our experiment sharp waves were determined in 21.4% (n=3) of CFL illuminated subjects and it is far above the mean populational percentage. Consequently, we could state about the external factor’s excitant effect on the brain.

There are several aspects of CFL lighting that may affect brain activity, including light spectrum specificities, flicker, electromagnetic radiation. CFL produce a different light spectrum from that of IL. The spectral power distribution of CFL is discrete due to a mix of phosphors on the inside of the tube, which each emit one color [26]. Mismatch to visual spectrum of the human eye may unsettle processes of neural calculation and modeling providing irregular color perception [27–28]. Flicker-associated problems are more concerned with traditional fluorescent strip lamps, which produce flickering at 50–60 Hz [29]. Although modulation of the light intensity could hardly be seen at 50 Hz, it becomes rather problematical for patients with migraine and traumatic brain injury due to lowered flicker fusion thresholds [9, 30–31]. “New generation” CFL use high-frequency electronic ballasts and do not flash [10]. Nevertheless, Berman et al. reported that higher frequency variation of luminoius intensity affects visual neurons. Such modulation excites rhythmic oscillations in human electroretinogram and is linked to eye strain, headaches and anxiety, though it is too rapid to be seen as a flicker [11, 14–15]. Furthermore, the high frequency currents generated to increase lamp efficiency can produce significant radiated noise and it becomes a matter of great concern [16, 32]. CFL emit EMF in the low (50 Hz) and high frequency (30–100 kHz) ranges [10, 32]. Although there is no doubt that short-term exposure to very high levels of electromagnetic fields (in the 100 kHz range) can be harmful to health, possible hazards of low level EMF remain disputable [32–35]. Substantial number of laboratory experiments, however, established that low frequency magnetic fields induce localized electric fields in the body [36–41]. Electric fields may interact with electrically excitable cells through voltage-gate ion channels and may excite nerve, muscle, and endocrine cells [42–46]. Moreover, some experiments confirm lower excitation threshold for neural network than for single neuron, suggesting CNS in vivo to be more likely sensitive to induced low frequency electric fields [44, 46–47]. This idea is also supported by recent studies [48–49]. In contrast, heart muscle tissue has electrically interconnected cells, thus weak electric fields are unlikely to have any effects on heart physiology [50]. Nevertheless, EMF could affect heart indirectly via vegetative nervous system [51]. Effects of low frequency EMF are also well determined on retinal function. Magnetic fields at 20–50 Hz are reported to induce visual sensations called magnetophosphens [52–53]. These phosphens appear as colorless flickering luminosities in the peripheries of visual fields, originating from interaction of induced electric fields with electrically sensitive retinal cells [54]. This phenomenon may be important in understanding EMF effects on neuronal network, particularly on cognitive processes as retina is a part of CNS and represents analogous processes [52, 55]. Several studies have investigated low frequency EMF impact on brain electrical activity. All studies differ in design, exposure conditions and detected changes which are most distinct in alpha band, though often discrepant [55–60]. Notwithstanding, Lyskov’s et al. detected increase in alpha power after exposure to EMF is rather similar to our results [58–59].

In addition, most CFL unsatisfied users describe a wide variety of nonspecific symptoms. Interestingly, these symptoms are similar to those reported by electrosensitive individuals. Since the lamps do not flash, it seems that pulsing EMF may take the main part affecting nervous system [29].

CONCLUSIONS

According to our study, it might be concluded that long-term exposure to CFL in comparison to incandescent light has different effects on brain electrical activity. Estimated higher prevalence of EEG alterations, as well as distinct alpha frequency and peak amplitude in CFL group, suggest irritant effects of CFL on the brain. CFL should not be used in EEG rooms not only because of interference with EEG electronics, but also due to provoked perturbations of brain electrical activity. Although CFL-related subjective and objective symptoms still remain uncertain, it becomes more obvious why some healthy persons and patients with
neurologic disorders develop various complaints in the presence of CFL. Further studies are needed to provide sufficient scientific evidence on health consequences connected to CFL explicating new and still unknown effects and potential risks.

Gauta:  Priimta spaudai:  
2010 10 23  2011 01 16

References
24. Thorner MW. Procurement of electroencephalographic tracings on 1,000 flying cadets for evaluating the Gibbs technique in relation to flying ability. Research Report No. 1, Project No. 7. The School of Aviation Medicine, 1942.

P. Areffjev, A. Chalkovskaja, R. Mamenikiené


Saunders RD, Jefferys JG. Weak electric field interactions in the central nervous system. Health Phys 2002; 83(3): 366–75.


