Original article

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Are Sub-Saharan epileptic people less photosensitive? A Senegalese study of photoparoxysmal response in a reference epilepsy centre*

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ABSTRACT – *Aims.* The photoparoxysmal response (PPR) is defined as the occurrence of generalized spike, spike-wave or polyspike-wave discharges consistently elicited by intermittent photic stimulation (IPS). PPR is not well studied in Sub-Saharan African people. We prospectively studied the epidemiological, clinical, and EEG characteristics of PPR among consecutive patients recorded at the clinical neurophysiology unit of Fann University Hospital (Dakar, Senegal). *Methods.* Among 6,808 EEG recordings including 3,065 pathological EEGs, we collected 56 EEGs with PPR (0.8% of all recorded EEGs and 1.8% of anormal EEGs), from 31 women and 25 men (sex ratio: 0.8). The mean age was 13.3 years (range: 8 months to 59 years). *Results.* The peak of photosensitivity was found in the range of 6 to 10 years. Of the PPR cases, 12 had clinical manifestations during IPS. Generalized epilepsy was diagnosed in 23 (41%) patients and 18 (32%) had focal epilepsies. The most epileptogenic stimulation frequencies were between 12 and 24 Hz (range: 1-28 Hz). PPR were mainly triggered during eye closure (64%), and 41 patients (73% of PPR cases) were classified as Type 4 (Waltz classification). *Conclusions.* Our results confirm lower rates of photosensitivity in African Sub-Saharan people compared with others. Although the current data do not support a role of short-term ambient light levels, subject to consistent data from larger cohorts, it may be interesting to study the probable epigenetic-mediated protective role of sunshine against photosensitivity.

Key words: epilepsy, photoparoxysmal response, intermittent photic stimulation

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Brain photosensitivity is an abnormal reactivity to specific photic stimuli. The phenomenon may be detected on the electroencephalogram (EEG) as visually-triggered epileptiform discharges, namely photoparoxysmal response (PPR) (Harding and Jeavons, 1994; Kasteleijn-Nolst Trenite *et al*., 2001). The clinical manifestations of this photosensitivity are seizures induced either by intermittent photic stimulation (IPS) or specific daily-life visual stimuli (such as flickering, light through trees, visual patterns, television, *etc*.) (Harding and Jeavons, 1994; Kasteleijn-Nolst Trenite *et al*., 2001). IPS protocols vary across epileptology teams, however, according to standardized IPS protocols, the patient is placed in a dim-light environment, 30 cm from the stroboscope (Kasteleijn-Nolst Trenite *et al*., 2013). Flash trains of an intensity of 1 joule (at least 0.70 joule), with increasing frequency (2 to 60 Hz), are emitted while the eyes are open, closed and during eye opening or closing (Kasteleijn-Nolst Trenité *et al*., 2012). The PPR usually appears between 10 and 30 Hz and eye closure is the most provocative condition (Kasteleijn-Nolst Trenite, 1989; Harding and Jeavons, 1994; Kasteleijn-Nolst Trenité *et al*., 2012). Photosensitivity may be asymptomatic or associated with idiopathic epilepsy in most cases (Ricci and Vigevano, 1993; Badinand-Hubert *et al*., 1998; Kasteleijn-Nolst Trenite *et al*., 2002). Photosensitive (PS) epilepsy is estimated to occur in approximately 1/4,000 of the population (Harding and Harding, 2010). It typically manifests around the age of puberty and disappears by the third decade depending on the underlying condition (Martins da Silva and Leal, 2017). Almost all the published studies show that women are more affected than men (Clement and Wallace, 1990; Obeid *et al*., 1991; Nagarajan *et al*., 2003).

Various factors (both environmental and genetic) have been postulated to play a crucial role in determining photosensitivity. This quest has led to some robust findings as well as some controversial reports (Fylan *et al*., 1999; Verrotti *et al*., 2012, 2005). However, currently, innate factors are considered as the main features correlated with photosensitivity (Verrotti *et al*., 2005; Kasteleijn-Nolst Trenite *et al*., 2013). Indeed, although there are some acquired conditions that may lead to photosensitivity, this reactivity is generally genetically determined (Waltz and Stephani, 2000).

Currently, photosensitivity has been studied in only few published African studies. Moreover, no West-African data are available, except for one 1983 study with major limitations (Danesi and Oni, 1983; Kasteleijn-Nolst Trenite *et al*., 2013; Martins da Silva and Leal, 2017). The main previous relevant African studies were completed in East-African and South-African countries, and were undeniably specific to anthropological and environmental factors associated with these areas, in contrast to the West-African population. Interestingly, during last decades, several studies have tried to find genetic and environmental factors that may support the epidemiological differences in PS epilepsy observed through the world. Therefore, our aim was to describe photosensitivity in patients with PS epilepsy in Senegal (West Africa) relative to other published data.

Methods and materials

Research location

The Neurology department of Fann University Hospital is a leading centre of neurological care for Senegal and neighbouring states. About 6,000-7,000 routine EEG recordings are performed every year, including both paediatric and adult patients.

Study population and data collection

For this cross-sectional study, we prospectively screened 6,808 consecutive outpatient routine EEGs recorded over one year. More than 99.5% of patients were black. In total, 3,065 individuals displayed epileptiform EEG discharges, including 56 individuals with PPR (*figure 1*). We then collected the following information from these PPR cases: demographic data (age, sex), age at epilepsy onset and seizure type (if relevant), daily-life photosensitivity history, family history of epilepsy and photosensitivity, risk factors for epilepsy, objective neurological examination findings, and EEG data (background and anomalies). We defined epileptic syndromes according to the revised International League Against Epilepsy classification (ILAE, 2017; Scheffer *et al*., 2017).

EEG recordings

All EEGs recordings were performed with an 18-channel EEG recording (Micromed, Brainquick amplificator SAM FC1 of 32 channels, Instruments Ltd., France) and standard 10-20 system (sampling rate: 256 Hz). Hyperventilation followed by IPS (3-5-min delay) was carried out for all 56 epilepsy patients. The IPS was performed using a photic stimulator (circular xenon lamp, central fixation point on diffuser, maximal intensity > 100 Nit-s per flash, enabling 1-60-Hz flashing range). The stroboscopic light source was placed at about 30 cm from the nasion. The room was kept dimly lighted during IPS. The sequence of IPS frequencies tested were 1, 4, 8, 12, 16, 18, 24 and 28 Hz (increasing and decreasing sequences), with eyes open, eye closure, and eyes closed. Note that patients had eyes

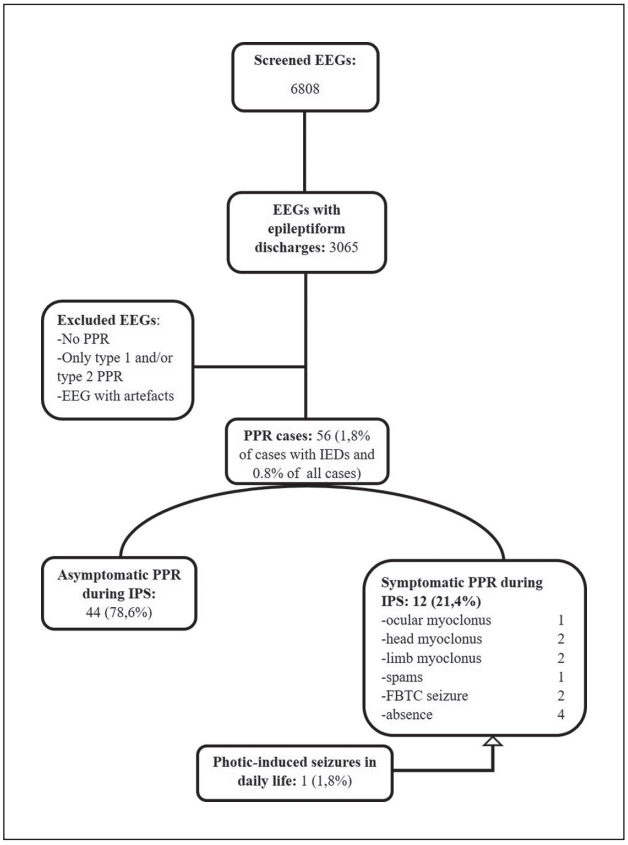

Figure 1. Patient selection flowchart.
PPR: photoparoxysmal response, IPS: intermittent photic stimulation, FBTC: focal to bilateral tonic-clonic.

open between stimulations and closed them at the start of stimulation. Only symptomatic PPRs led to IPS cessation.

Definition of PPR

PPR were analysed according to the criteria proposed by Waltz *et al*. (Waltz *et al*., 1992) (*table 1*). We collected cases with pathological PPR based on the occurrence of:
– any PPR Type 3 and 4 with clinical history of epilepsy;
– or any epileptic seizure triggered by IPS.
Waltz Type 1 and 2 PPRs were excluded because of the poor inter-rater reliability, the uncertain semiological value and the fact that these responses are usually excluded in PPR studies.

Results

Demographic characteristics

We collected 56 EEGs (all from different patients) with PPR (0.8% of a total of 6,808 EEGs recorded; 1.8% of 3,065 pathological EEGs) (*figure 1*), from 31 women and

Table 1. Waltz classification (Verrotti *et al*., 2012; Waltz *et al*., 1992).

Type 1	Spikes within an occipital rhythm
Type 2	Parieto-occipital spikes with a biphasic slow wave
Type 3	Parieto-occipital spikes with a biphasic slow wave and spread to the frontal region
Type 4	Generalized discharges with spikes and waves or polyspikes and waves

25 men (sex ratio of 0.8). The mean age was 13.3 ± 10.2 years (range: 0.8-59) and 42 patients (64.6%) were under 20 years old (*figure 2*). Among the 56 patients, 16 epilepsy-known cases were referred for control EEG, 33 for suspected epilepsy, six for unexplained cognitive impairment, and one for unexplained headaches.

Epileptiform findings and epilepsy diagnosis

Based on spontaneous interictal EEG findings and clinical follow-up, epilepsy was diagnosed in all the 56 patients. Of them, 40 were not known to be epileptic. During the recordings, 12 patients showed clinical manifestations triggered by IPS. Only one complained of photosensitive seizures triggered by television and sunlight, and this patient had a PPR during IPS without associated clinical manifestation. Overall, we diagnosed generalized epilepsy in 23 patients (41%), focal epilepsies in 18 (32%), combined generalized and focal epilepsies in 10 (18%), and unknown onset in five (9%). The distribution of patients according to the ILAE epilepsy classification (2017) is shown in *table 2*.

Photic reactivity

Among the 56 patients, 41 (73%) displayed Type 4 PPR (*figures 3, 4*) while the others displayed Type 3 response during IPS (*samples are illustrated in figure 5*). The PPR was mostly observed in the patients with idiopathic generalized epilepsy. The frequencies that most commonly elicited PPR ranged between 12 and 24 Hz (*figure 6*). The PPR outlasted the IPS by more than 100 ms in eight patients (14.2%). Photoconvulsive response in the form of jerks involving the limbs or whole body, or eyelid twitching, occurred in 12 patients (21.4%). Eye closure PPR was the most provocative stimulus (*figure 6*).

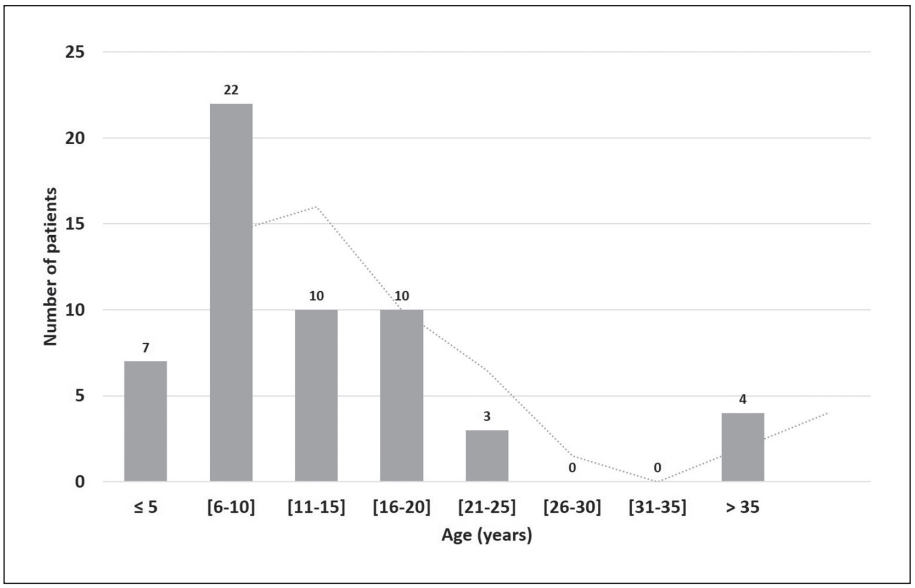

Figure 2. Age repartition of patients with photoparoxysmal response. Most of the PPR cases were 6-10 years old and 75% of the patients were 6-20 years old.

Table 2. The distribution of patients according to epilepsy classification (ILAE, 2017).

Epilepsy diagnosis	Number of patients	Total
Focal onset	Motor onset (*n* = 17)	18 (32%)
	Non-motor onset (*n* = 1)	
Generalized onset	Motor (*n* = 16) *(4 cases of juvenile myoclonic epilepsy)*	23 (41%)
	Non-motor (absence) Typical (*n* = 5) Atypical (*n* = 1) Eyelid myoclonia (*n* = 1)	
Unknown onset	Motor (*n* = 4)	5 (9%)
	Non-motor (*n* = 1)	
Combined generalized and focal	N/A	10 (18%)

N/A: not applicable.

Discussion

Photosensitivity is one of the well-studied phenomena in epileptology. Historically, the variation observed in initial studies worldwide oriented numerous subsequent investigations in an attempt to identify factors and mechanisms underlying the features of this abnormal brain reactivity. Currently, interesting findings have been widely published. However, there is a lack of data describing PS epilepsy features in some geographic areas, mainly African regions. In our prospective study, we aimed to describe photosensitivity in Senegal (West Africa) by analysing clinical and EEG findings in order to identify local specificities, if any. We collected 56 EEGs with generalized PPR out of 3,065 pathological EEGs (a total of 6,808 routine EEGs were performed for outpatients regardless of the clinical indication) during 2016. The prevalence of photosensitivity in the present study is among the lowest (0.8% of all EEGs and 1.8% of EEGs with spontaneous epileptiform discharges) compared with data in the literature (de Graaf, 1992; de Graaf *et al*., 1995; Lu *et al*., 2008; Verrotti *et al*., 2012; Kasteleijn-Nolst Trenite *et al*., 2013; Koutroumanidis *et al*., 2015; Whitehead *et al*., 2016). Numerous prevalence studies have been conducted in various countries. However, interpretation of these results is complicated by the significant variation of study designs with respect to race, age (children versus adults), indication of EEGs included (confirmed epilepsy versus suspected epilepsy versus any indication), IPS frequencies used, and definition of PPR. The most relevant studies are summarized in *table 3* with respect to these parameters. Nevertheless, in Holland, Germany, and South Africa, PPR is found in 5-8% of epileptic patients (Kasteleijn-Nolst Trenite, 1989; de Graaf, 1992; Nagarajan *et al*., 2003), compared to only 1-2% in India, Nigeria, or Zimbabwe (Danesi and Oni, 1983; Saleem *et al*., 1994; Familusi *et al*., 1998). The lower prevalence of PPR among epilepsy patients

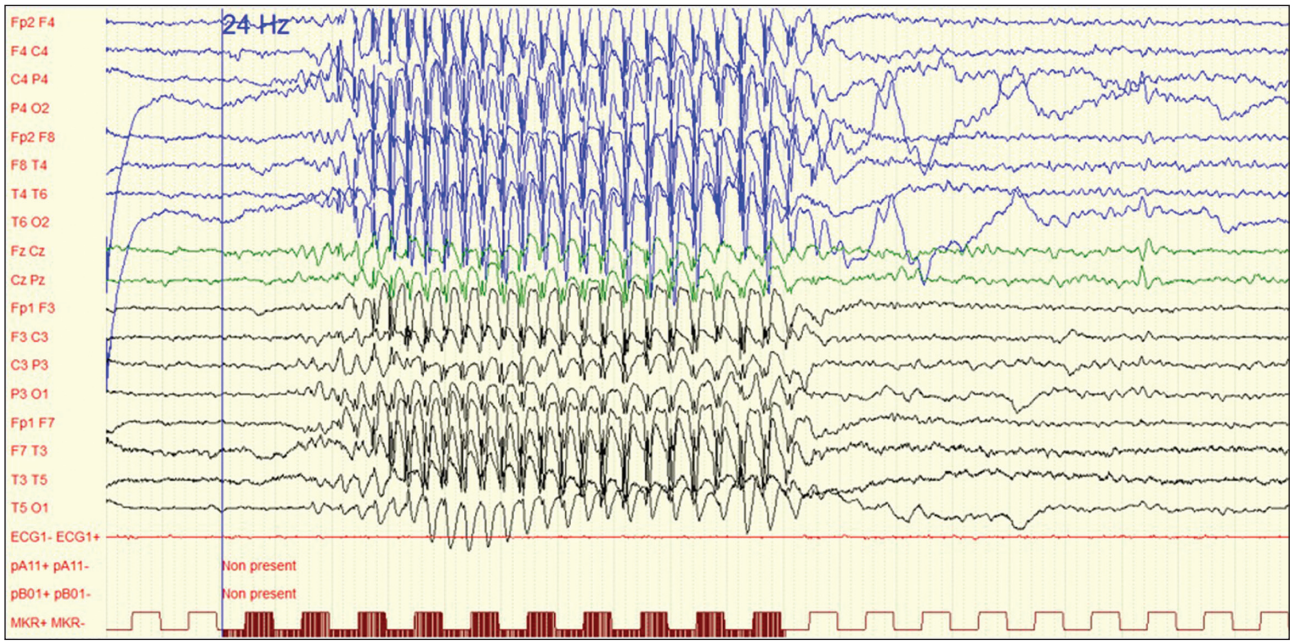

Figure 3. Example of Type 4 PPR with a fragmented build-up. Case 2: A 22-year-old epileptic patient known to have epilepsy, investigated for bilateral tonic-clonic seizures evolving over two years; medication: phenobarbital 100 mg die. Fragmented generalized bursts of polyspike-wave discharges are shown during IPS at: (A) 4 Hz; (B) 12 Hz; and (C) 16 Hz. (high filters: 70 Hz; low filters 0.530 Hz; notch filter 50 Hz; sensitivity: 100 mV/cm; speed: 25 mm/s).

in our study could be partly due to the high level of sunshine throughout the year in tropical African regions, which may be speculated to have a protective influence on IPS. In this regard, Danesi reported seasonal variations in the incidence of PPR among photosensitive epileptic patients (Danesi, 1988). In his study, 49 white patients were recorded through the seasons: 22 in summer, 35 in winter, 17 in spring and 16 in autumn. The lowest incidence of PPR occurred among summer recordings (9.1%) and the highest among winter recordings (96%). Moreover, 11 patients (seven with generalized epilepsies and four focal cases) were recorded both during winter and summer. In these patients, only 2/14 recordings showed PPR in summer whereas only 1/11 recordings was free of PPR during winter. However, following this publication, in a short letter, a subsequent response was specifically addressed by Trenite *et al*. (1989) who reported opposite findings and conclusions (Trenite *et al*., 1989). In this report, there were no significant seasonal differences in the incidence of photosensitivity with regards to the relationship between new identified PS patients and number of new EEG referrals per season (20/586 in spring, 20/492 in summer, 37/609 in autumn and 23/655 in winter; total of 2,342). Furthermore, when dividing their PS cohort into two equal groups based on their degree of photosensitivity at the time of first EEG, no seasonal effect was found. Although they used a different methodology, these findings raise questions about the impact of duration of exposure to global sunlight. In any case, the extrinsic hypothesis, with an emphasis on individual life-time exposure to sunshine, is not well supported by several studies targeting ethnic differences in people living in the same geographic areas. Indeed, Familusi *et al*. (1998) found lower PPR prevalence in black people among 9,082 Zimbabwean patients of 0-25 years old referred for routine EEG (Familusi *et al*., 1998). They reported a PPR prevalence of 0.2% in black people, but 2.1% in Caucasian and Asian individuals (Familusi *et al*., 1998). Moreover, in this study, 0.6% of Métis people showed PPR, thus suggesting genetic inheritance. This was similar to the results of De Graaf *et al*. (1992) who found PPR in 0.4% of black people, 4% in Métis people and 5.2% in Caucasians (de Graaf, 1992). De Graaf *et al*. confirmed this tendency in 1995, reporting PPR in 2.7% Caucasians (72/2657), 0.1% black people (1/848) and 0.9% Métis people (55/5,958) (de Graaf *et al*., 1995). Although these studies suggest a robust ethnicity-related genetic element to PPR, an epigenetic impact of ambient sunshine cannot be excluded. This hypothesis may explain why Indian people (mostly living in tropical areas) seem to have lower PPR prevalence than other Asians such as Japanese and Chinese (Saleem *et al*., 1994; Shiraishi *et al*., 2001; Bai *et al*., 2019).

The main age range of our PS cohort was 6-10 years (22/56 patients) followed by 10-20 years (20/56 patients). Thus, 87.5% of the patients with PPR were under 20 years old while 75% of them were 6-20 years old.

Table 3. Comparative table of PPR prevalence in different studies.

Country	Study design	No. of EEGs (no. of PPRs)	PPR definition (No. of PPR cases)	Mean age (range)	Ethnic PPR prevalence (%)	Provoking IPS frequency * (whole range) in Hz	Reference
Africa							
Namibia	Retrospective study, consecutive patients with suspected epilepsy	1493 (35)	Generalized pSpk, S-W, pS-W activity, elicited by IPS, not frequency-locked to the stimulus and outlasting it by at least 100 msec	Not provided, but 51.7% were 6-25y	Black: 3/806 (0.4) White: 15/361 (4.2) Mixed: 17/326 (5.2)	Black: 7-31.3 Mixed: 12.9-31.5 White: 13.6- 32.5 (1-40)	(de Graaf, 1992)
South Africa	Retrospective study, consecutive patients with presumed diagnosis of epilepsy (excluded: history of febrile convulsions, acute symptomatic epilepsy, and history of alcoholism)	15292 (128)	Generalized pSpk, S-W, pS-W activity, elicited by IPS, not frequency-locked to the stimulus and outlasting it by at least 100 msec	25.7 (0 to more than 35y)	White: 72/2657 (2.7) Mixed: 55/5958 (0.9) Black: 1/ 848 (0.1)	White: mean: 12.3 ± 6.0 to 29.0 ± 9.1 Mixed: mean: 13.2 ± 4.8 to 27.5 ± 7.1 Black: NA (1-40)	(de Graaf *et al.*, 1995)
Nigeria	Prospective study, 362 consecutive epileptic patients, and 102 consecutive nonepileptic patients	362 (6) 102 (0)	Photoconvulsive responses to IPS (not clearly defined in the text)	19.2y (0.3-70y)	Black: 6/362 (1.6)	10-20 (1-30)	(Danesi and Oni, 1983)
Senegal	Prospective study, consecutively referred for routine EEG (mainly epilepsy)	6808 (56)	(A) any PPR Type 3 and 4 based on Waltz *et al* classification with clinical history of epilepsy; or (B) any epileptic seizure triggered by IPS (56)	13,3y (0.8-59y)	Black: 56/6808 (0.8)	Most provoking: 12-24 (1-28)	Present study

Table 3. Comparative table of PPR prevalence in different studies (*continued*).

Country	Study design	No. of EEGs (no. of PPRs)	PPR definition (No. of PPR cases)	Mean age (range)	Ethnic PPR prevalence (%)	Provoking IPS frequency * (whole range) in Hz	Reference
Asia							
Saudi Arabia	Prospective study, 327 consecutive patients with confirmed epilepsy, and 192 non-epileptic patients	327 (24)	Generalized and synchronous S-W (or pS-W), involving the anterior and posterior regions, and usually persisting after stimulus cessation	M: 26.2 ± 11.38 y (15-80 y) F: 25.9 ± 10.3 (15-70 y)	Arabic: - 24/327 (7.3) for PWE - 0/192 for non-epileptic patients	NA (1-50)	(Obeid *et al.*, 1991)
North India	Prospective study, consecutive patients with confirmed epilepsy, referred for routine EEG (excluded: single seizure, febrile seizure, sibling of PS patients, unexpected PPR in non-epileptic patient)	1000 (6)	Generalized Spk, S-W, pS-W, consistently elicited by IPS, not frequency-locked to the stimulus and outlasting the stimulus train by at least 100 ms	16.8 ± 11.85 y (1 to 60+ y)	Indian: 6/1000 (0.6)	Most provoking: 6-30 (6-60)	(Saleem *et al.*, 1994)
South India	Retrospective study, patients with confirmed diagnosis of epilepsy	575 (20)	Generalized Spk, S-W or pS-W paroxysm at least twice during the same frequency of IPS, irrespective of duration of the paroxysm or whether it outlasted the IPS or not. Excluded: isolated occipital spikes and generalized or focal bursts of slowing without Spk.	20.9 ± 12.4 (0.5-66)	Indian: 20/575 (3.5%)	Most provoking: 18 (1-24)	(Radhakrishnan *et al.*, 1998)

Table 3. Comparative table of PPR prevalence in different studies (*continued*).

Country	Study design	No. of EEGs (no. of PPRs)	PPR definition (No. of PPR cases)	Mean age (range)	Ethnic PPR prevalence (%)	Provoking IPS frequency * (whole range) in Hz	Reference
China	Prospective study, consecutive patients with suspected epilepsy	5482 (73)	Patients with generalized epileptic discharges and localized epileptic discharges during IPS	15 y (0.3-79y)	Chinese: 73/5482 (1.3)	8-25 (1-60)	(Bai *et al.*, 2019)
Japan	Retrospective study, consecutive patients with suspected epilepsy	2187 (37)	Bisynchronous diffuse S-W and multiple S-W, not frequency-locked to the stimulus and outlasting the stimulus train by 100 ms (37)	24.2 y (1-81 y)	Japanese: 37/2187 (1.7)	NA (6-33)	(Shiraishi *et al.*, 2001)
Europe							
Germany	Retrospective study, consecutive patients with confirmed epilepsy	1044 (103)	Precipitation of a clinical seizure or S-W, pS-W or repetitive Spk of a frequency independent of the flash rate (103)	14.4y (10-25y)	White +++: 103/1044 (9.9)	NA (3-30 Hz)	(Wolf and Goosses, 1986)
UK	Prospective study, all patients admitted for routine EEG recording	5384, including 4420 PWE (79)	Unequivocal generalized epileptiform interictal EEG activity (*i.e.* Waltz Type 3 or 4 PPR) NOT seen in the resting recording (76)	30y (1-99y)	White +++: 79/5384 (1.5)	NA	(Whitehead *et al.*, 2016)

Table 3. Comparative table of PPR prevalence in different studies (*continued*).

Country	Study design	No. of EEGs (no. of PPRs)	PPR definition (No. of PPR cases)	Mean age (range)	Ethnic PPR prevalence (%)	Provoking IPS frequency * (whole range) in Hz	Reference
America							
Brasil	Prospective study, clinically "normal" children and adolescents (excluding enuresis or encopresis, prematurity, sleep abnormalities, first-degree relatives with epilepsy and seizures)	510	NA	(6 - 18 y)	NA (1.4)	NA	(Kasteleijn-Nolst Trenite et al., 2003)
US/ Boston	Consecutive suspected epileptic patients	3557 (553)	Generalized Spk, pSpk or sharp wave burst or a generalized spike-wave paroxysm (553)	Infants to 80 y	NA:27/553 (4.9)	NA	(Jayakar and Chiappa, 1990)
Oceania							
Australia	Retrospective study, consecutive children who underwent "EEG + IPS"	263	Waltz Type 1-4 PPR	(5-15)	NA	10-15 Hz (1-30 Hz)	(Nagarajan *et al.*, 2003)

PPR: photoparoxysmal response; IPS: intermittent photic stimulation; Spk: spike; pSpk: polyspike; S-W: spike and wave; pS-W: polyspike and wave; NA: not available. +++mostly (i.e. exact statistics are unknown).
* the methodology for determining the most inductive frequencies of PPR varies among studies, therefore the data provided in the table on these frequencies are for illustrative purposes only and not for strict comparison.

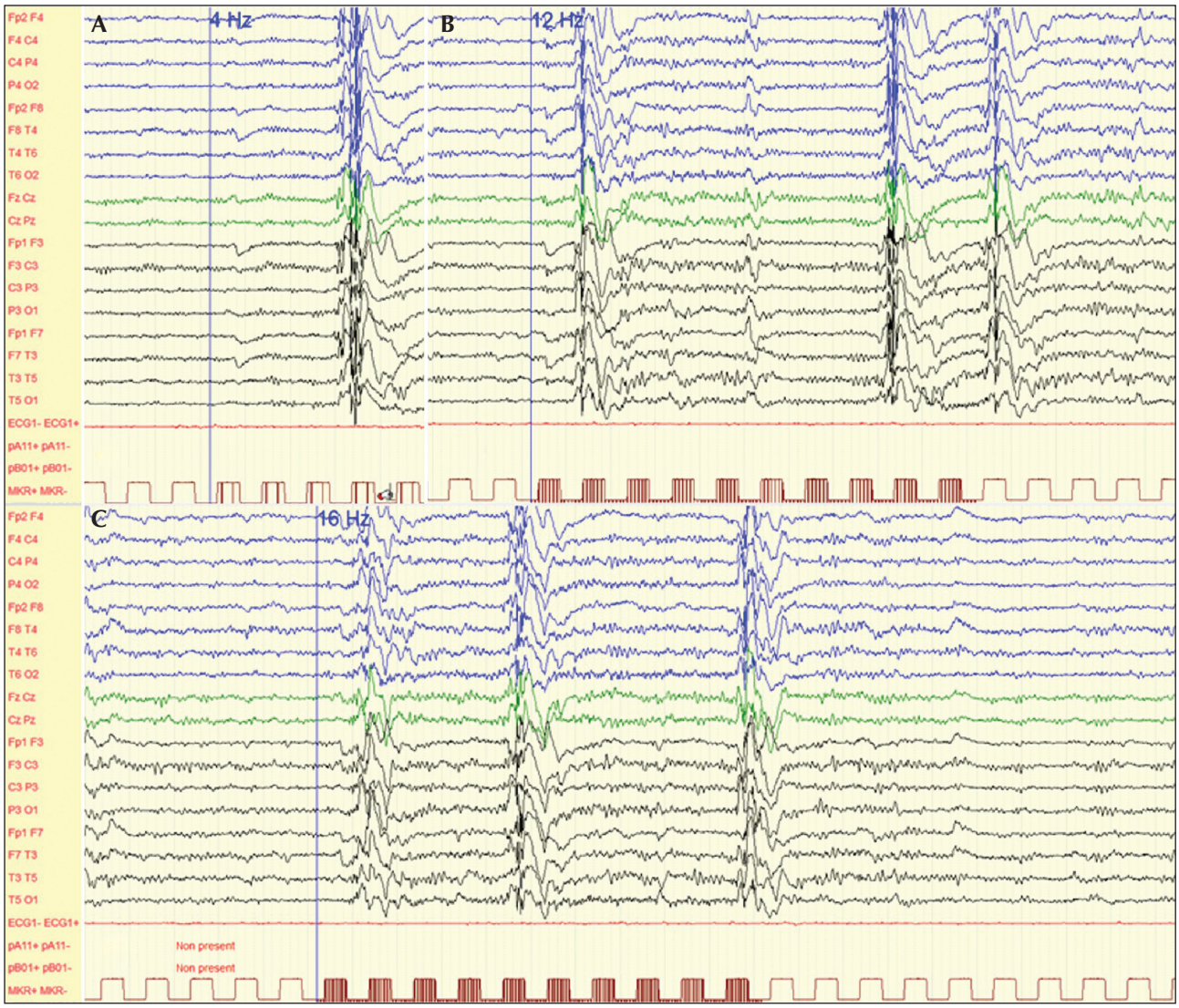

Figure 4. Example of Type 3 PPR. Case 3: A 41-year-old patient with cognitive impairment and seizures since childhood under valproic acid. Polyspike-wave discharges in parieto-occipital regions with anterior spreading are shown, recorded at: (A) 4 Hz, and (B) 12 Hz. (high filters: 70 Hz; low filters 0.530 Hz; notch filter 50 Hz; sensitivity: 100 mV/cm; speed: 25 mm/s).

Several authors claim that maximum photosensitivity is mainly observed around puberty (Kasteleijn-Nolst Trenite, 1989; Clement and Wallace, 1990). In addition, PS is reputed to significantly decline after 15-20 years old (Verrotti *et al*., 2012; Kasteleijn-Nolst Trenite *et al*., 2013). Our findings are consistent with this age-PS relationship. Regarding sex repartition, our cohort with PPR included 31 women and 25 men (an approximate 2:1 ratio). Similarly, previous prevalence studies of PPR and visually-induced seizures also showed a clear predominance of about 60-70% in females for children, adolescents and adults (Clement and Wallace, 1990; Obeid *et al*., 1991; Familusi *et al*., 1998; Nagarajan *et al*., 2003; Verrotti *et al*., 2012). However, there are studies suggesting that within some age groups, boys are more photosensitive than girls (Kasteleijn-Nolst Trenite, 1989).

The most epileptogenic IPS frequencies were between 8 and 24 Hz with peaks at 12 and 24 Hz. Harding and Jeavons found similar results, namely at 10-25 Hz (Harding and Jeavons, 1994). Regarding eye conditions, it is largely accepted that eye closure is by far the most sensitive state (Kasteleijn-Nolst Trenite, 1989; Kasteleijn-Nolst Trenité *et al*., 2012). Our results showed that most photosensitive subjects experienced PPR during eye closure on command. In a study by Kasteleijn-Nolst-Trenite *et al*., out of 100 photosensitive patients based on IPS, 93% had discharges at eye closure, 81% with eyes closed and 66% with eyes open (Kasteleijn-Nolst Trenite *et al*., 2002). The hypothesis

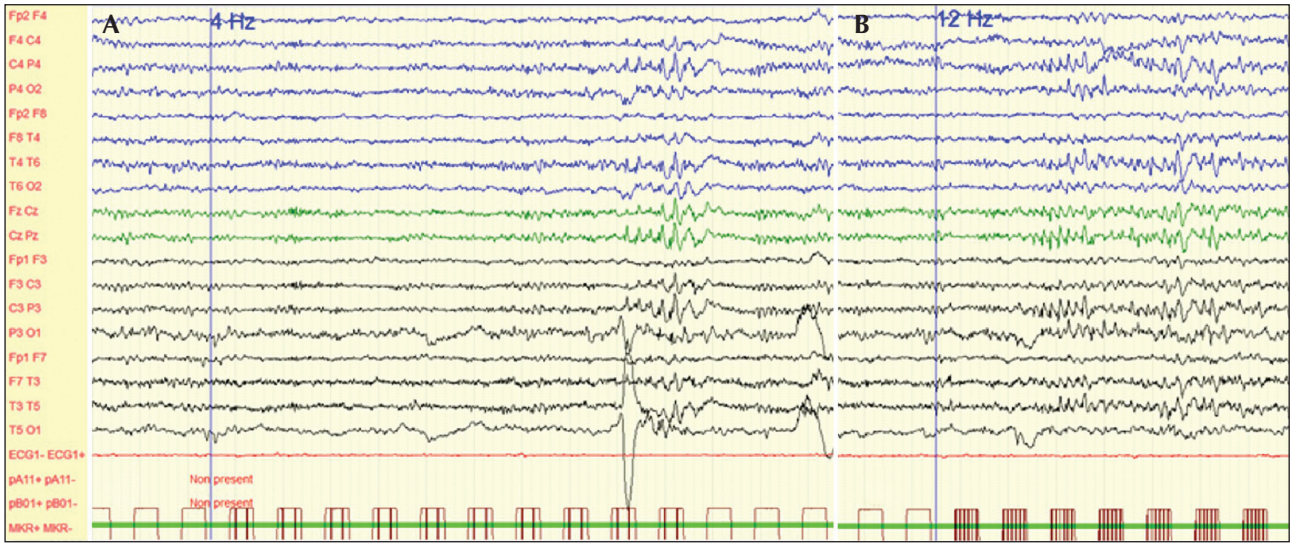

Figure 5. Reactivity to intermittent photic stimulation (IPS) frequencies in patients who showed PPR. PPR appeared with eyes open in only one case. The condition that most frequently resulted in PPR in response to IPS was eye closure. Data relative to eyes open are not included as this was minimized during the recordings of this study.

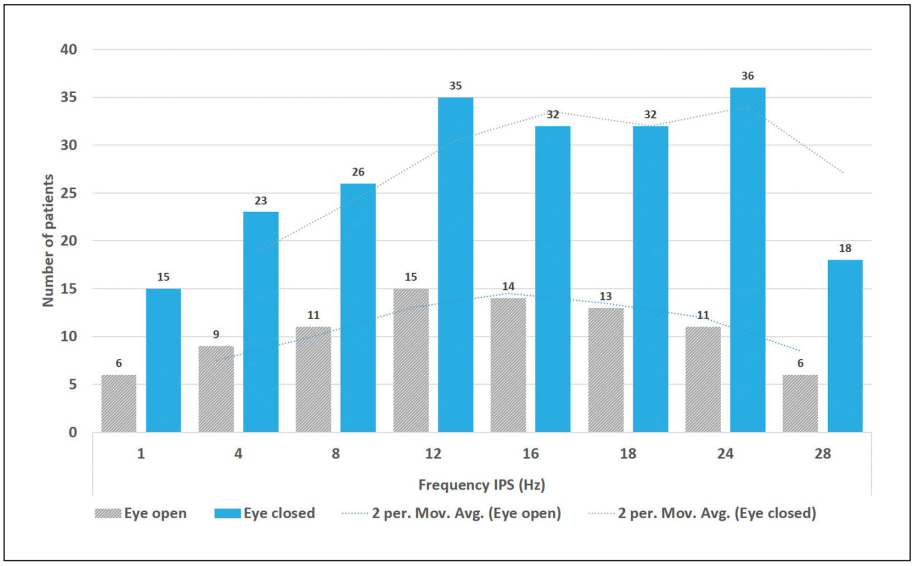

Figure 6. Example of Type 4 PPR. Case 1: A 16-year-old female patient with PPR consisting of generalized spike- and polyspike-wave discharges at 3.5 Hz (IPS at 24 Hz). (high filters: 70 Hz; low filters 0.530 Hz; notch filter 50 Hz; sensitivity: 100 mV/cm; speed: 25 mm/s).

that this is due solely to the extent of illumination of the retina is debatable. Other factors may be associated; the motor effect of eye closure or loss of attention and visual fixation (Wilkins *et al.*, 2004).

In all our 56 patients with Type 3-4 PPR, an epilepsy had been diagnosed. This is disappointing since numerous studies confirmed that PPR prevalence in non-epileptic patients ranges from 0.5 to 8.9% (Quirk *et al.*, 1995; Verrotti *et al.*, 2012). However, the present study included only EEG laboratory referrals with a high threshold for medical indication. This potentially reduced the probability of recruiting non-epileptic PS patients. On the other hand, we could hypothesize that selecting only Type 3-4 PPR is also a factor that reduces the chance of identifying epilepsy-free PPR. However, in this regard, Waltz *et al.* (1992), studying 108 PS probands and associated 114 relatives (siblings), reported that 76.3% had Type 3 and 4 PPRs among 93 who were non-epileptic. Thus, elective Type 3-4 PPR inclusion is insufficient to explain

100% epilepsy diagnosis. The remaining hypothesis is that Type 3-4 PPRs are frequently associated with epilepsy diagnosis in Senegalese people. Regarding epilepsy type, approximately 32% of our PPR cases were associated with focal epilepsy and 10% with combined generalized and focal epilepsy. This finding is consistent with a predominant range of 20-40% reported in the literature worldwide (total range of 2-65% to the best of our knowledge) (Verrotti *et al*., 2012; Kasteleijn-Nolst Trenite *et al*., 2017). Of the 56 PPR cases recorded, 12 (21.4%) showed clinical manifestations during IPS. One other, who was complaining of photosensitive events triggered by television and sunlight, had an asymptomatic PPR during IPS. In comparison, Kasteleijn-Trenite *et al*. reported clinical symptoms or signs in 75% of cases based on a series of 36 PS patients (Kasteleijn-Nolst Trenite *et al*., 1987). However, all the 36 patients were selected with the high-threshold criterion of generalized PPR outlasting the photic stimulus. In another UK multicentric study, about 49% of 79 PPRs were associated with epileptic clinical manifestations, regardless of the timing of PPR (5,383 patients in total). These two studies show higher clinical reactivity in PS patients compared with our cohort. This may be a continuum of the higher PS rates in Caucasian people.

Conclusion

We found a PPR prevalence of about 0.8% in all the patients referred for routine EEGs and in 1.8% of those who showed epileptiform discharges. Although further work is needed, it appears that genetic factors, but not high sunlight levels, are the main determinants of photosensitivity. Above all, our findings corroborate that African people are less photosensitive compared with Caucasians. The surprising absence of non-epileptic patients among those who displayed generalized PPRs suggests that Type 3-4 PPRs are frequently associated with a diagnosis of epilepsy in Senegalese people (and probably anthropological relatives) compared with others. There are still many unresolved issues, in terms of physiopathology, classification, and genetics, especially in black Africans, and further knowledge on this subject would be enriched by multicentric studies in Africa. □

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TEST YOURSELF

(1) What was the prevalence of photosensitivity in this Senegalese study?

(2) What were the most epileptogenic frequencies?

(3) Are Sub-Saharan Africans less photosensitive than others?

Note: Reading the manuscript provides an answer to all questions. Correct answers may be accessed on the website, www.epilepticdisorders.com, under the section "The EpiCentre".