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Afternystagmus in darkness after suppression of optokinetic nystagmus: an interaction of motion aftereffect and retinal afterimages

Chen, C C ; Huang, M Y Y ; Weber, K P ; Straumann, D ; Bockisch, C J

Abstract: The afternystagmus that occurs in the dark after gaze fixation during optokinetic stimulation is directed in the opposite direction relative to the previous optokinetic stimulus. The mechanism responsible for such afternystagmus after suppression of optokinetic nystagmus (ASOKN) is unclear. Several hypotheses have been put forward to explain it, but none is conclusive. We hypothesized that ASOKN is driven by the interaction of two mechanisms: (1) motion-aftereffect (MAE)-induced eye movements and (2) retinal afterimages (RAIs) produced by fixation during the suppression of optokinetic nystagmus (OKN). We examined the correlation among ASOKN, MAE-induced eye movements, and RAIs in healthy subjects. Adapting stimuli consisted of moving random dot patterns and a fixation spot and their brightness was adjusted to induce different RAI durations. Test patterns were a stationary random dot pattern (to test for the presence of a MAE), a dim homogeneous background (to test for MAE driven eye movements), and a black background (to test for ASOKN and RAIs). MAEs were reported by 16 out of 17 subjects, but only 7 out of 17 subjects demonstrated MAE-induced eye movements. Importantly, ASOKN was only found when these seven subjects reported a RAI after suppression of OKN. Moreover, the duration of ASOKN was longer for high-brightness stimuli compared with low-brightness stimuli, just as RAIs persist longer with increasing brightness. We conclude that ASOKN results from the interaction of MAE-induced eye movements and RAIs.

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1 **Afternystagmus in darkness after suppression of optokinetic** 2 **nystagmus: an interaction of motion aftereffect and retinal** 3 **afterimages**

4
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17 18 **Abstract**

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20 in the opposite direction relative to the previous optokinetic stimulus. The mechanism responsible for
21 such afternystagmus after suppression of optokinetic nystagmus (ASOKN) is unclear. Several
22 hypotheses have been put forward to explain it, but none is conclusive. We hypothesized that ASOKN
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25 nystagmus. We examined the correlation among ASOKN, MAE-induced eye movements, and RAIs in
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28 dot pattern (to test for the presence of a MAE), a dim homogeneous background (to test for MAE
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30 by 16 out of 17 subjects, but only 7 out of 17 subjects demonstrated MAE-induced eye movements.
31 Importantly, ASOKN was only found when these 7 subjects reported a RAI after suppression of
32 optokinetic nystagmus. Moreover, the duration of ASOKN was longer for high-brightness stimuli
33 compared to low brightness stimuli, just as RAIs persist longer with increasing brightness. We
34 conclude that ASOKN results from the interaction of MAE-induced eye movements and RAIs.

35 **Keywords:** Afternystagmus after suppression of optokinetic nystagmus (ASOKN) ∙ Motion aftereffect
36 (MAE) ∙ Motion-aftereffect-induced eye movements ∙ Retinal afterimage (RAI) ∙ Human
37

1

2 **Introduction**

3 Optokinetic nystagmus (OKN), which is induced by a constantly moving large-field visual surround,
 4 consists of slow-phase eye movements to minimize retinal slip velocity and fast-phase eye movements
 5 for position reset. When the optokinetic stimulus is replaced by total darkness, nystagmus continues
 6 with a gradual decay (Ohm 1921). Optokinetic afternystagmus (OKAN) is thought to be generated by a
 7 mechanism that stores slow-phase eye velocity signals during optokinetic stimulation and keeps
 8 releasing the eye velocity signal for a certain period after cessation of the optokinetic stimulus. This so-
 9 called velocity storage mechanism (VSM) stores velocity signals from the optokinetic and vestibular
 10 systems to preserve their low frequencies and hence enhance visual-vestibular cooperation during
 11 rotation in the light (Cohen et al. 1977; Raphan et al. 1977; Raphan et al. 1979; Laurens and Angelaki
 12 2011).

13 A different afternystagmus in total darkness occurs after optokinetic stimulation, during which
 14 subjects suppress OKN by fixating their eyes upon a space-fixed visual target. This afternystagmus
 15 after suppression of OKN (in the following abbreviated with ASOKN) is directed opposite of the
 16 previous optokinetic stimulus (Korenaga et al. 1996), which is in contrast to OKAN, during which the
 17 nystagmus occurs in the same direction. Kudo et al. (2002) suggested that this “reverse OKAN” results
 18 from the VSM, which is charged by retinal slip (a sensory signal) and then generates an eye velocity
 19 signal in the opposite direction of optokinetic stimulation to counteract the optokinetic signal. This
 20 keeps the fovea on a stationary target in the presence of the moving large-field visual surround. In a
 21 different interpretation, Ventre-Dominey and Luyat (2009) suggested that ASOKN is linked to the
 22 vestibular and pursuit motor systems rather than a purely sensory signal (retinal slip). Note, however,
 23 that ASOKN has not always been found; e.g. Fletcher et al. (1990) found that fixation of a small target
 24 during optokinetic stimulation almost completely prevented the development of afternystagmus and
 25 thus concluded that gaze fixation actively prevents storage of visual signals.

26 Gaze fixation upon a space-fixed target during optokinetic stimulation can induce a perceptual
 27 motion aftereffect (MAE). The perceptual MAE is an illusory motion perception: a stationary stimulus
 28 is perceived to move in the opposite direction of the previous optokinetic stimulus (Anstis et al. 1998;
 29 Mather et al. 1998). The perceptual MAE may drive eyes to pursue the illusory motion. Watamaniuk
 30 and Heinen (2007) demonstrated that after gaze fixation during unidirectional optokinetic stimulation
 31 eye movements were biased in the opposite direction. In the same study, these authors showed that
 32 such MAE-induced eye movements do not occur in the dark, i.e. only appear in the presence of a visual
 33 input (they used a moving random dot stimulus). This is analogous to the perceptual MAE, which also
 34 requires a visual stimulus (Wohlgemuth 1911; Spiegel 1960; Verstraten et al. 1994; Thompson and
 35 Wright 1994).

36 Prolonged ocular fixation of a visual target may induce retinal afterimages (RAIs) (Brown,
 37 1965). RAIs, which occur on the retina due to adaptation of photoreceptors, are optical illusions that
 38 persist after the exposure to the original image (Shimojo et al. 2001). It has been demonstrated that the
 39 duration of RAIs is positively correlated with the intensity and lasting time of the original image
 40 (Granit et al. 1930; Feinbloom 1938; Nagamata 1951; Lu et al. 2006). Thus, during optokinetic

stimulation, ocular fixation of a space-fixed visual target may induce a RAI as well.

We hypothesized that ASOKN is caused by an interaction of MAE-induced eye movements and RAIs and not the result of any VSM. Although MAE-induced eye movements depend on visual input and should not be activated in the dark, RAIs can be an alternative visual source to elicit these eye movements. Consequently, such eye movements would be directed opposite of the previous optokinetic stimulus. Moreover, since RAIs are eye-fixed, they would move with the MAE-induced movements of the eyes, thereby stimulating further eye movements. Thus, the interaction of MAE-induced eye movements and RAIs could form a positive feedback loop to keep the eyes moving, i.e. nystagmus, in the direction of the MAE-induced eye movements as long as RAIs are present. Such a mechanism can maintain nystagmus independent of the VSM.

To test our hypothesis, we did a correlation study among MAE-induced eye movements, RAIs, and ASOKN in healthy human subjects. Moreover, visual stimuli of different brightness were used to evoke different RAI durations. According to our hypothesis, ASOKN would only occur in those subjects who demonstrate MAE-induced eye movements and perceive a RAI. In addition, the duration of ASOKN should be longer after higher brightness of the stimulus, just as RAIs persist longer with increasing brightness.

Materials and Methods

Human subjects

Experiments were performed on 10 males and 7 females, aged 23-49 years, with no abnormal neurologic or ophthalmologic history. Visual acuity was normal or corrected-to-normal. All subjects gave their informed written consent and the experiment was approved by the local ethics committee.

Experimental setup

A head-mounted video-oculography (VOG) device (EyeSeeCam, Munich, German) running at a frame rate of 220 Hz was used for the eye-movement recording. Subjects faced the center of a screen which was placed 100 cm away from the head of subject. The screen was 178 cm in width and 130 cm in height, which hence covered 80° of the horizontal visual field and 66° of the vertical visual field. A digital projector (Panasonic PT-AE7000 Projector), operating at 60 frames per second and a spatial resolution of 1920 x 1080 pixels, was used. A custom-developed script in MATLAB (Mathworks, Natick, MA, USA) and its Psychophysics Toolbox extensions (Brainard 1997; Pelli 1997; Kleiner et al. 2007) were applied to control visual stimuli. Eye position was calibrated at $\pm 10^\circ$ of the center of the screen with a custom-developed script.

Visual stimulation

The optokinetic stimulus was a random pattern of 600 white dots (3 deg^2) with a dot lifetime of 150 milliseconds. The speed of each dot was 30 deg/s toward the right. In conditions with gaze fixation, a space-fixed 1.6 deg^2 yellow dot with a black ring inside was projected on the center of the screen (Fig. 1). Low and high brightness conditions were used to evoke different retinal afterimage (RAI) durations. In the high-brightness condition, the luminance of the moving dots and the yellow fixation dot were 330 lux and 314 lux, respectively. In the low brightness condition, the luminance of the

1 moving dots and the yellow fixation dot were 68 lux and 70 lux.

2 Experimental conditions

3 Six conditions were applied in this study (Fig. 1). The sequence of conditions was randomized in each
4 subject. Each condition was presented to subjects once.

5 Conditions 1 to 4 started with a 3-minute period of suppression of optokinetic nystagmus (OKN)
6 and were followed by a 1-minute task. During the period of suppression of OKN, subjects were asked
7 to fixate their eyes upon the yellow stationary dot in the center of the screen.

8 Condition 1 tested whether or not the perceptual motion aftereffect (MAE) occurred after
9 suppression of OKN. After suppression of OKN, the speed of each random dot was set to zero during
10 the 1-minute task. In other words, each random dot just stayed in the same position for 150
11 milliseconds and then reappeared in another position. The central yellow fixation dot remained
12 unchanged. Subjects were asked to report any percept of motion in the random dot pattern while
13 fixating the central yellow dot. The stimuli used in condition 1 were of high brightness.

14 With condition 2 we tested whether or not MAE-induced eye movements occurred. The period of
15 suppression of OKN was the same as in condition 1. After suppression of OKN, no structured pattern
16 was present on the screen. Thus, the screen luminance (0.17 lux) was uniform across the whole screen.
17 Such a visual environment provides a visual input without fixation targets to evoke possible MAE-
18 induced eye movements.

19 Conditions 3 (low brightness) and 4 (high brightness) were applied to test how afternystagmus
20 after suppression of OKN (ASOKN), if present, changed with the RAI duration. After suppression of
21 OKN, the visual surround was switched to dark. The dark condition was always examined before
22 Conditions 3 and 4 by asking subjects whether they saw any light while they were covered by a
23 lightproof cloth. Since the light from the LCD beamer we used could not be turned completely off, we
24 found that using the cloth to completely cover the subjects head was a fast and effective way to create a
25 dark environment for the subject. Afterwards, subjects were asked if they saw a RAI during the last
26 minute.

27 Conditions 5 and 6 were applied to test whether or not low and high brightness optokinetic
28 stimuli could evoke the same degree of optokinetic response (OKR). No fixation was provided in these
29 two conditions. The low brightness condition was used in Condition 5 and the high brightness
30 condition was used in Condition 6.

31 Data analysis

32 The movement of the left eye was analyzed. Data analysis was all done by a custom-developed
33 program written in MATLAB. Eye position was smoothened by a Gaussian low-pass filter with a cut-
34 off frequency of 18 Hz. Eye velocity was derived from the smoothed position signal. Blinks were
35 deleted by visual inspection. Fast-phase selection was done by (1) dividing the whole eye position
36 curve into segments based on the eye-movement direction and (2) identifying a segment as a saccade if
37 its maximum velocity was > 30 deg/s. The other segments with a maximum velocity < 30 deg/s would
38 be identified as part of slow phase. The duration of ASOKN and MAE-induced eye movements was
39 determined by the number and direction of fast phase. Nystagmus was identified by the fast phases

continuously moving towards one direction more than 5 times. The duration was counted from the first fast phase to the last one. For instance, eye movements shown in Fig. 2A, Condition 2, would be recognized as nystagmus because the eye movements consisted of 8 fast phases moving uninterruptedly in one direction and the duration from the first fast phase to the last one was approximately 9 seconds. Maximum slow-phase eye velocities were calculated by searching for the maximum eye velocity in the opposite direction of the fast phases. Statistical tests (paired one-tailed t-test) were done in MATLAB with the Statistics Toolbox.

Results

In Conditions 1 to 4, all subjects were able to maintain gaze fixation while a random dot pattern was moving in the background (see Fig. 2). In all subjects, the absolute median eye velocity was < 1 deg/s during the period of suppression of optokinetic nystagmus (OKN). Conditions 5 (low brightness) and 6 (high brightness) tested whether or not low and high brightness optokinetic stimuli would evoke similar slow phase eye velocity during steady-state OKN. The average median eye velocity in Condition 5 (low brightness) was 17.6 ± 8.1 deg/s while the same value in Condition 6 (high brightness) was 19.0 ± 7.4 deg/s; and the difference was not significant (paired one-tailed t-test, $t = 1.61$, $p = 0.1272$).

Perceptual motion aftereffect

After suppression of OKN, 16 out of 17 subjects reported that the space-fixed random dot pattern, in which each random dot stayed in the same position for 150 milliseconds and then reappeared in other positions, was perceived to move towards the left (Table 1). Since all subjects were asked to stare at the yellow fixed dot after suppression of OKN, gaze remained stable (see Fig. 2ABC, Condition 1 for examples).

Motion-aftereffect-induced eye movements

Whether or not MAE-induced eye movements appeared in subjects was evaluated in Condition 2. After suppression of OKN, 7 out of 17 subjects generated a nystagmus in the opposite direction of the previous optokinetic stimulation as they faced the stationary screen with uniformly distributed dim light (example: Fig. 2A, Condition 2), while no nystagmus was recorded in the others (example: Fig. 2B, Condition 2).

Retinal afterimage (RAI)

Subjects were asked whether RAIs appeared after suppression of OKN in Conditions 3 (low brightness) and 4 (high brightness). Eleven subjects reported that a RAI appeared after the low brightness condition (Condition 3) while 15 subjects saw a RAI after the high brightness condition (Condition 4). Ten of 11 subjects who saw a RAI in both conditions reported that the RAI after the high brightness condition lasted longer than after the low brightness condition, while 1 subject reported no difference.

Afternystagmus after suppression of optokinetic nystagmus (ASOKN)

Whether ASOKN requires the presence of RAI was tested in Conditions 3 (low brightness) and 4 (high brightness). Ten subjects did not generate ASOKN in either condition (see Fig. 2B). Two subjects had

ASOKN only in Condition 4, while 5 subjects had it in both conditions (see Table 1). Duration and maximum slow-phase velocity of ASOKN between the two conditions were compared to determine the influence of stimulus brightness on ASOKN. The duration of ASOKN was significantly longer in the high-brightness condition (paired one-tailed t-test, $t = 2.69$, $p = 0.0361$). However, no significant difference was found between the maximum slow phase velocities of ASOKN in Conditions 3 and 4 (paired one-tailed t-test, $t = 1.85$, $p = 0.1125$).

Interestingly, all subjects who had ASOKN in Condition 4 demonstrated MAE-induced eye movements in Condition 2. Two subjects, who showed MAE-induced eye movements in Condition 2 and reported no RAI in Condition 3, did not generate ASOKN in Condition 3 (Table 1).

Discussion

We investigated the mechanism responsible for afternystagmus after suppression of optokinetic nystagmus (ASOKN), in which slow-phase eye movements are in the opposite direction of previous optokinetic stimulation. This ASOKN was only found in a minority of healthy subjects tested. Five out of 17 subjects (29%) showed ASOKN after presenting the low-brightness stimuli, while 7 out of 17 subjects (41%) showed ASOKN after presenting the high-brightness stimuli. These percentages are less than in a previous study that reported ASOKN in 14 out of 23 subjects (61 %) (Ventre-Dominey and Luyat 2009, Table 2). In contrast, Fletcher et al. (1990) reported that none of their 4 subjects (0 %) generated ASOKN in the opposite direction of the preceding optokinetic stimulus. Hence, it seems that ASOKN highly depends on the individual subject and the parameters of the visual stimulation, such as brightness.

We found that ASOKN can be elicited if a subject shows motion-aftereffect-induced (MAE-induced) eye movements and also reports seeing retinal afterimages (RAIs). Specifically, the subjects who showed MAE-induced eye movements in Condition 2 and saw a RAI in Conditions 3 or 4 generated ASOKN (see Table 1). In the remaining subjects, no ASOKN was recorded. The notion that RAIs are the essential driver of ASOKN was underlined by the fact that the high-brightness stimuli induced a longer duration of ASOKN than the low-brightness stimuli (Fig. 3a), as RAIs persist longer with increasing brightness (Granit et al. 1930; Feinbloom 1938; Nagamata 1951).

The concurrence of RAIs, MAE-induced eye movements, and ASOKN in individual subjects suggests that ASOKN results from an interaction of MAE-induced eye movements and RAIs. The MAE-induced eye movements are very similar to ASOKN in respect to the eye-movement direction and the precondition, i.e. gaze fixation during optokinetic stimulation. But, the MAE-induced eye movements require visual input (Watamaniuk and Heinen 2007), different to the condition of testing ASOKN, where the visual surround is totally dark (Kudo et al., 2002; Ventre-Dominey and Luyat, 2009). However, RAIs, if also present after suppression of optokinetic nystagmus (OKN), can be the visual input for the MAE-induced eye movements. Moreover, the eye-fixed RAIs that move with the MAE-induced eye movements of the eyes may further stimulate eye movements. Such an interaction between the MAE-induced eye movements and RAIs can induce an afternystagmus in the dark after suppression of OKN, i.e. ASOKN. Either RAIs or MAE-induced eye movements alone are not sufficient to generate ASOKN.

Perceptual motion aftereffect (MAE) and the MAE-induced eye movements

In this study, 16 out of 17 subjects (94%) reported a perceptual MAE in Condition 1, but only 7 out of 17 subjects (41%) generated MAE-induced eye movements in Condition 2 (see Table 1). Why did not all subjects with a perceptual MAE show MAE-induced eye movements? We speculate that, in these subjects, the perceptual MAE was not strong enough to drive the eyes to follow the motion illusion. Further study is needed.

Afterimages

We controlled the brightness of the space-fixed yellow dot and the random dot pattern, to induce different strengths of RAIs. Twelve out of 17 subjects (71%) saw a RAI in Condition 3 (low brightness), while 16 out of 17 subjects (94%) reported it appeared in Condition 4 (high brightness). Moreover, 11 out of 12 subjects who saw a RAI in Conditions 3 and 4 reported that the RAI duration was longer and stronger after the high brightness condition. Our results agree with previous studies, in which brightness of the image positively correlated to the RAI duration (Granit et al. 1930; Feinbloom 1938; Nagamata 1951; Lu et al. 2006).

Velocity storage mechanism

Previous studies suggested that ASOKN may be related to some sort of velocity storage mechanism (VSM), which is charged by optokinetic signals during the period of suppression of OKN and then causes afternystagmus in the subsequent dark (Kudo et al., 2002; Ventre-Dominey and Luyat, 2009). However, Fletcher et al. (1990) suggested that gaze fixation prevents the coupling of optokinetic signals into the VSM. Our study supports the conclusion that it is not the VSM being charged by retinal slip during fixation of a stationary target. In our experimental data, Conditions 5 and 6, which used the same moving random dot pattern, except for the brightness, evoked similar degrees of OKN (see Table 1). In other words, although the brightness was different, the two optokinetic stimuli caused a similar amount of retinal slip. In Conditions 3 and 4, however, we found that the high and low brightness conditions significantly influenced the duration of ASOKN (see Fig. 3A); high brightness evoked a longer ASOKN. This phenomenon cannot be well explained by a hypothesis involving a VSM.

Summary

In summary, we found that ASOKN occurred only if subjects had the MAE-induced eye movements and saw a RAI after suppression of OKN. Our results therefore suggest that ASOKN may be the result of an interaction of MAE-induced eye movements and RAIs.

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9 **Legend**

10 **Fig. 1** Experimental conditions. Each row represents the stimuli in a condition. The left column shows
11 the motion stimuli (of different brightness), with or without a central fixation dot during the first
12 adaptation phase of each condition. The right column represents the stimuli in the testing phase.
13 Condition 1 was applied to test the perceptual MAE. Dots of the random dot pattern in the testing
14 phase did not move. Condition 2 was applied to test the MAE-induced eye movements. Conditions 3
15 and 4 were designed to investigate how stimulus brightness affects ASOKN. Conditions 5 and 6, in
16 which there was no central fixation spot, were designed to investigate how stimulus brightness affects
17 the optokinetic response. The high brightness visual stimuli were applied on Conditions 1, 2, 4, and 6
18 while the low brightness ones were applied on Conditions 3 and 5 (see the “visual stimulation” section
19 of Methods for the details of the stimulus brightness).

20 **Fig. 2** Typical examples of eye movements in (A) Subject 1 and (B) Subject 13. Condition number and
21 name are shown on the left side of each eye position trace. **A** Subject 1 held gaze stable during the
22 period of suppression of OKN in Conditions 1 to 4. In Condition 1, gaze stayed stable in the last
23 minute due to the strong fixation stimulus. The subjects reported a perceptual MAE. In Condition 2, the
24 MAE-induced eye movements were found after suppression of OKN. In Conditions 3 and 4, ASOKN
25 was found after suppression of OKN. **B** Subject 13 also held gaze stable during the period of
26 suppression of OKN in Conditions 1 to 4. In Condition 1, the subject reported a perceptual MAE. In
27 Conditions 2–4, no eye movement was found after suppression of OKN. Slow phases (velocity <30
28 deg/s) are marked in grey, and fast phases are marked in black.

29 **Fig. 3** Duration (A) and maximum slow phase eye velocity (B) of ASOKN in those subjects who had
30 ASOKN in either Conditions 3 or 4.

31 **Table 1** Summary of experimental data. Numbers at the top of the table correspond to the condition
32 number. Subjects reporting a perceptual MAE after Condition 1 were marked with “√”. In Conditions
33 2–4, the median eye velocity of the first 20 seconds after the period of suppressed OKN was calculated.
34 Subjects reporting a RAI in Conditions 3 and 4 were marked with a “√”. Moreover, maximum slow
35 phase velocity (SPV) and duration of the MAE-induced eye movements and ASOKN were calculated
36 only in the subjects who had a nystagmus after suppression of OKN. In Conditions 5 and 6, median eye
37 velocity during the 3-minute optokinetic stimulation was calculated. The number in the last row
38 indicates the number of subjects who reported or had perceptual MAE, MAE-induced eye movements,
39 RAIs, and ASOKN. The shaded area indicates the subjects who demonstrated the MAE-induced eye
40 movements in Condition 2.

Subject	1	2			3								5	6
	Perceptual MAE	MAE-induced eye movements			Afternystagmus after suppression of OKN								OKN	
	High brightness	High brightness			Low brightness				High brightness				Low brightness	High brightness
		Median Velocity (°/s)	Maximum SPV (°/s)	Duration (s)	Retinal Afterimage	Median Velocity (°/s)	Maximum SPV (°/s)	Duration (s)	Retinal Afterimage	Median Velocity (°/s)	Maximum SPV (°/s)	Duration (s)	Median velocity (°/s)	Median velocity (°/s)
1	√	-2.4	10	9	√	-1.9	10	10	√	-4.6	12	45	23.4	24.8
2	√	-2.5	15	60	√	-3	15	32	√	-3.5	23	42	28	29
3	√	-0.7	7	8	√	-0.6	7	4	√	-0.9	4	6	6	12.6
4	√	-2	8	50	√	-1.8	5	15	√	-2.1	9	40	24	22
5	√	-2.3	10	16	√	-3.1	8	18	√	-3.4	8	18	25.6	27.4
6	√	-1.6	10	22	X	0.3	X	X	√	-0.5	4	8	23.4	23.5
7	√	-0.8	3	11	X	-0.4	X	X	√	-0.8	2	10	12.7	10
8	√	-0.4	X	X	√	0.3	X	X	√	0	X	X	27.2	29
9	√	0.4	X	X	√	0.2	X	X	√	0.4	X	X	20.3	22.9
10	√	-0.3	X	X	X	0.3	X	X	√	-0.1	X	X	25	23
11	√	-0.4	X	X	√	-0.2	X	X	√	-0.1	X	X	19	15
12	√	-0.1	X	X	√	-0.2	X	X	√	-0.3	X	X	16	21.4
13	√	-0.1	X	X	√	-0.2	X	X	√	0	X	X	9.7	12.3
14	√	-0.1	X	X	√	-0.1	X	X	√	0.4	X	X	4.2	5.7
15	√	-0.2	X	X	X	-0.3	X	X	X	-0.2	X	X	6.5	16.3
16	√	0.1	X	X	X	-0.1	X	X	X	0	X	X	19.7	20.8
17	X	-0.3	X	X	X	-0.4	X	X	√	-0.3	X	X	8.7	7
# of presence	16	7			11	5			15	7			17	17

√=present; X=absent

Figure 1

Figure 2

A

B

Figure 3