Clinical characteristics and mortality risk in relation to obstructive and central sleep apnoea in community-dwelling elderly individuals: a 7-year follow-up

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Abstract

Background: little is known about demographic and clinical characteristics associated with sleep-disordered breathing (SDB) and obstructive sleep apnoea (OSA) or central sleep apnoea (CSA) in community-dwelling elderly. We also examined these (OSA and CSA) associations to all-cause and cardiovascular (CV) mortality.

Methods: a total of 331 community-dwelling elderly aged 71–87 years underwent a clinical examination and one-night polygraphic recordings in their homes. Mortality data were collected after seven years.

Results: a total of 55% had SDB, 38% had OSA and 17% had CSA. Compared with those with no SDB and OSA, more participants with CSA had a left ventricular ejection fraction <50% (LVEF <50%) ischaemic heart disease (IHD) and transient ischaemic attack (TIA)/stroke. There was no difference in the rate of IHD and TIA/stroke between OSA and no SDB, but more LVEF <50% was found in those with OSA. CSA significantly increased the risk for all-cause (P = 0.002) and CV mortality (P = 0.018) by more than two times. After adjustments for CV disease, diabetes and the biomarker NT-pro-brain natriuretic peptide CSA associations to all-cause mortality and CV mortality lost significance.

Conclusion: OSA, in persons >75 years does not appear to be associated with cardiovascular disease (CVD) disease or mortality, whereas CSA might be a pathological marker of CVD and impaired systolic function associated with higher mortality.

Keywords: elderly, obstructive sleep apnoea, central sleep apnoea, mortality
Background

Health-care professionals working night-shifts often notice that sleep-disordered breathing (SDB), including obstructive sleep apnoea (OSA) and central sleep apnoea (CSA), occurs among elderly patients. OSA is characterised by a cessation in airflow (apnoea) or reduced airflow (hypopnoea) for at least 10 s because of complete or partial upper airway obstruction during sleep accompanied with maintained, increased or paradoxical respiratory effort in a response to generate airflow [1]. In contrast to OSA, CSA involves reduced or no respiratory efforts during the apnoeic/hypopnoeic episode [1, 2]. The incidence of OSA increases with age; in middle-aged men and women 9 and 4%, respectively [3], have OSA compared with 24 and 16% of men and women aged 60–70 years [4]. Less is known about the prevalence of CSA in the general population [1], but in a study performed in men only, the prevalence rose from 0% in those 20–44 years compared with 12% in those aged 65–100 years [5].

Despite the fact that OSA and CSA are common among the elderly, most studies concerning prevalence, demographic and clinical characteristics and prognosis have focused on healthy middle-aged populations. OSA and CSA cause mechanical, haemodynamic and neurohormonal changes that contribute to stress of the heart [6]. OSA in CSA cause mechanical, haemodynamic and neurohormonal changes that contribute to stress of the heart [6]. OSA and CSA in elderly patients. OSA is characterised by a cessation in airflow (apnoea) or reduced airflow (hypopnoea) for at least 10 s because of complete or partial upper airway obstruction during sleep accompanied with maintained, increased or paradoxical respiratory effort in a response to generate airflow [1]. In contrast to OSA, CSA involves reduced or no respiratory efforts during the apnoeic/hypopnoeic episode [1, 2]. The incidence of OSA increases with age; in middle-aged men and women 9 and 4%, respectively [3], have OSA compared with 24 and 16% of men and women aged 60–70 years [4]. Less is known about the prevalence of CSA in the general population [1], but in a study performed in men only, the prevalence rose from 0% in those 20–44 years compared with 12% in those aged 65–100 years [5].

The aims of the present study were (i) to describe demographic and clinical characteristics associated with OSA or CSA, and (ii) to examine whether OSA or CSA is associated with all-cause or CV mortality within 7 years observation in community-dwelling elderly.

Materials and methods

Participants

All subjects were chosen from a cohort that was evaluated in a previous study which took place between 1998 and 2001 [13]. Then, all inhabitants aged 65–82 years who were living in a rural municipality in the southeast of Sweden were included. In total, 1,130 persons were invited to participate. Of those, 876 agreed to participate (participation rate 78%). The present study started in January 2003 when the 876 persons previously examined were asked to participate in a new study. A total of 675 were included in the present study. Reasons for being excluded were deaths (n = 102), had moved to nursing homes or left the area (n = 29) or declined (n = 70). All 675 subjects met a cardiologist for a clinical examination, and, out of these 346 (51%) also agreed to undergo sleep respiratory recordings. Those participating in the sleep study did not differ from those who did not, with the exception of more history of respiratory disease (asthma or chronic obstructive pulmonary disease) (17 versus 12%, P = 0.04) in sleep study participants. The study protocol was approved by regional ethical review board, Linköping, Sweden.

Clinical examination, co-morbidities and current treatment

Every participant was examined by an experienced cardiologist who took the patient history and performed a clinical examination. The LVEF was determined by echocardiography. Normal systolic function corresponded to an LVEF ≥50%, whereas impaired systolic function corresponded to an LVEF <50%. N-terminal fragment of pro-brain natriuretic peptide (NT-pro-BNP) was drawn with the patient in sitting after an overnight fasting and resting for 30 min. NT-pro-BNP was measured using an electrochemiluminescence immunoassay (Elecsys 2010, Roche Diagnostics, Mannheim, Germany). Blood pressure was measured in supine position after fasting overnight. Systolic and diastolic blood pressures were used to calculate the mean arterial blood pressure (MAP) [(1/3 × systolic blood pressure + 2/3 × diastolic blood pressure)]. Diabetes mellitus was defined as ongoing treatment or fasting blood glucose ≥7 mmol/l. Hypertension was defined as a previous diagnosis, or blood pressure of more than 140/90 mmHg. Ischaemic heart disease (IHD) was defined as a history of angina pectoris, and/or myocardial infarction. Transient ischaemic attack/stroke (TIA/stroke) was defined by history. A respiratory disease was established whether the participant had a diagnosis, or was undergoing treatment for chronic pulmonary disease or asthma.

Sleep study

The sleep studies were performed during one night in the patients’ homes with polygraphic equipment (Embletta, Flaga Medical, Inc., Reykjavik, Iceland). The recording included airflow measured by a nasal pressure cannula, posture, physical motions, abdominal and thoracic movements and finger pulse oximetry. Apnoeas, hypopnoeas and SDB severity were scored according to the American Academy of Sleep Medicine Task Force guideline [1]. No SDB was defined as an apnoea-hypopnoea index (AHI) AHI <5, mild SDB was defined as an AHI 5–15 and moderate severe SDB as AHI ≥15. Apnoeas was characterised as either obstructive or central based on the persistence of continued abdominal/thoracic movements or not. Participants were considered to have OSA or CSA whether they had AHI ≥5 and more than 50% of their apnoeas were obstructive or central, respectively. The start and end of the analysis period were set according to the combination of information provided by the participants (sleep-log and interview), tracing quality and breathing pattern. Sleep scorings were done by the main author (P.J.).
Table 1. Comparison of no SDB, OSA and CSA on demographic and clinical characteristics

<table>
<thead>
<tr>
<th></th>
<th>No SDB, n = 148</th>
<th>OSA, n = 123</th>
<th>CSA, n = 54 (17%)</th>
<th>No SDB versus OSA</th>
<th>No SDB versus CSA</th>
<th>CSA versus CSA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean (SD)</td>
<td>78 (3.5)</td>
<td>78 (2.7)</td>
<td>78 (3.3)</td>
<td>0.35</td>
<td>0.98</td>
<td>0.5</td>
</tr>
<tr>
<td>Male sex, % (n)</td>
<td>43 (64)</td>
<td>46 (57)</td>
<td>72 (39)</td>
<td>0.61</td>
<td>&lt;0.001</td>
<td>0.001</td>
</tr>
<tr>
<td>Body mass index, mean (SD)</td>
<td>26.7 (3.6)</td>
<td>28.1 (4.9)</td>
<td>27.9 (3.7)</td>
<td>0.06</td>
<td>0.035</td>
<td>0.93</td>
</tr>
<tr>
<td>MAP, mean (SD)</td>
<td>98.7 (10.2)</td>
<td>99.6 (11.1)</td>
<td>97.8 (10.8)</td>
<td>0.48</td>
<td>0.6</td>
<td>0.33</td>
</tr>
<tr>
<td>NT-pro-BNP, mean (SD)</td>
<td>491 (797)</td>
<td>425 (557)</td>
<td>762 (962)</td>
<td>0.13</td>
<td>0.01</td>
<td>0.001</td>
</tr>
<tr>
<td>LVEF &lt;50%, % (n)</td>
<td>7 (11)</td>
<td>19 (23)</td>
<td>37 (20)</td>
<td>0.005</td>
<td>&lt;0.001</td>
<td>0.009</td>
</tr>
<tr>
<td>AH1, mean (SD)</td>
<td>2.0 (1.2)</td>
<td>15.6 (9.9)</td>
<td>18.2 (12.3)</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>0.52</td>
</tr>
<tr>
<td>ODI, mean (SD)</td>
<td>1.8 (1.2)</td>
<td>13.4 (9.6)</td>
<td>15.6 (11.2)</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>0.34</td>
</tr>
<tr>
<td>CAI, mean (SD)</td>
<td>0.2 (0.4)</td>
<td>0.9 (1.4)</td>
<td>8.3 (9.3)</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>OAI, mean (SD)</td>
<td>0.5 (0.7)</td>
<td>5.9 (6.3)</td>
<td>1.1 (1.7)</td>
<td>&lt;0.001</td>
<td>0.004</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>SaO2, mean (SD)</td>
<td>94.8 (1.7)</td>
<td>93.9 (1.8)</td>
<td>94.1 (1.2)</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>0.77</td>
</tr>
<tr>
<td>Diabetes, % (n)</td>
<td>22 (32)</td>
<td>21 (26)</td>
<td>28 (15)</td>
<td>0.32</td>
<td>0.36</td>
<td>0.34</td>
</tr>
<tr>
<td>Hypertension, % (n)</td>
<td>71 (105)</td>
<td>73 (90)</td>
<td>76 (41)</td>
<td>0.68</td>
<td>0.48</td>
<td>0.7</td>
</tr>
<tr>
<td>IHD, % (n)</td>
<td>21 (31)</td>
<td>23 (26)</td>
<td>43 (23)</td>
<td>0.72</td>
<td>0.002</td>
<td>0.007</td>
</tr>
<tr>
<td>TIA/stroke, % (n)</td>
<td>5 (8)</td>
<td>7 (9)</td>
<td>24 (13)</td>
<td>0.52</td>
<td>&lt;0.001</td>
<td>0.002</td>
</tr>
<tr>
<td>Respiratory disease, % (n)</td>
<td>18 (27)</td>
<td>15 (19)</td>
<td>13 (7)</td>
<td>0.34</td>
<td>0.38</td>
<td>0.67</td>
</tr>
<tr>
<td>ACEI/ARB, % (n)</td>
<td>21 (31)</td>
<td>30 (37)</td>
<td>35 (19)</td>
<td>0.08</td>
<td>0.038</td>
<td>0.5</td>
</tr>
<tr>
<td>β-Blockers, % (n)</td>
<td>27 (40)</td>
<td>42 (52)</td>
<td>57 (31)</td>
<td>0.008</td>
<td>&lt;0.001</td>
<td>0.06</td>
</tr>
<tr>
<td>Diuretics, % (n)</td>
<td>32 (48)</td>
<td>37 (45)</td>
<td>39 (21)</td>
<td>0.47</td>
<td>0.39</td>
<td>0.77</td>
</tr>
<tr>
<td>Hypnotics, % (n)</td>
<td>16 (23)</td>
<td>15 (18)</td>
<td>9 (5)</td>
<td>0.83</td>
<td>&lt;0.001</td>
<td>0.23</td>
</tr>
</tbody>
</table>

Bolded P-values are considered as significant after correction for Bonferroni (0.05/(n = 3 – 1), P = 0.025).

ACEI/ARB, angiotensin-converting inhibitor/angiotensin receptor blockers; AH1, apnoea-hypopnoea index; BMI, body mass index; CAI, central apnoea index; CSA, central sleep apnoea; IHD, ischaemic heart disease; IVEF, left ventricular ejection fraction; MAP, mean arterial blood pressure; NT-pro-BNP, N-terminal fragment brain natriuretic peptide; OAI, obstructive apnoea index; ODI, oxygen desaturation index; SaO2, oxygen saturation; SDB, sleep-disordered breathing; TIA/stroke, transient ischaemic attack/stroke.

Mortality

All mortality was registered during the 7-year follow-up period and was collected from the National Board of Health and Welfare, Stockholm, Sweden and from autopsy records. CV mortality was defined as deaths caused by HF and/or fatal arrhythmias, or by IHD, or sudden deaths or cerebrovascular deaths. No patients were lost during the follow-up period.

Statistical analysis

Continuous variables were analysed with Student’s t-test or Mann–Whitney U test, depending on distribution of normality, whereas categorical variables were analysed with Chi-square tests. Testing of multiple comparisons was corrected with the Bonferroni method. Survival curves for all-cause and CV mortality were obtained by Kaplan–Meier analysis and Log-rank estimations. Cox proportional regression hazard analysis was used to evaluate whether CSA was a potential predictor of all-cause and CV mortality. During the study period 61 deaths were registered and of these 35 were of CV causes. In survival analyses, the number of deaths is the limiting sample size and we choose to calculate with 15 deaths per covariate [14]. In addition to the predictor CSA, in the Cox-regression analyses three more covariates were added. Variables presented in Table 1 associated with mortality were; age; NT-pro-BNP; LVEF <50%; diabetes; IHD and TIA/stroke. Age and the LVEF <50% were both found associated with NT-pro-BNP and therefore eliminated. We chose NT-pro-BNP because it is found to be sensitive to subtle alterations in left ventricular filling pressures [15] as well as a strong independent predictor for the prognosis in community-dwelling elderly [16]. IHD and a TIA/stroke were amalgamated into one group labelled cardiovascular disease (CVD). Hence, in addition to predominant CSA, diabetes, CVD and NT-pro-BNP were used as covariates in the Cox-regression analyses. A P < 0.05 was considered significant. Statistics were processed with the SPSS version 18.0 (SPSS, Inc., IBM, Chicago, IL, USA).

Results

Of the 346 sleep recordings, 15 were lost due to technical failure. In six of the participants, two females and four males, the type of SDB was not possible to determine and were excluded. The final study population in this study consists of 325 persons.

Clinical characteristics of predominant OSA and CSA

SDB (i.e. AH1 ≥5) was found in 55%, 38% had OSA and 17% had CSA. Characteristics, co-morbidities and medications for those with no SDB, OSA and CSA are presented in Table 1. There were significantly more males found in the group with CSA (72%) both compared with those with OSA (57%, P = 0.001) and to those with no SDB (43%, P < 0.001). Body mass index (BMI), was higher in those
with CSA compared with the group no SDB, but this difference was not significant after correction for multiple comparisons. Impaired systolic function was associated with SDB. In those with CSA 37% was found to have an LVEF <50%. This was significantly different compared with the 7% (P < 0.001) and 19% (P = 0.009) found in those with no SDB or OSA. Those with OSA had more of the LVEF <50% (P = 0.005) compared with those with no

**Table 2.** Cox-regression models analysing the association between participants with CSA and all-cause mortality during 7-year follow-up

<table>
<thead>
<tr>
<th></th>
<th>All-cause mortality, hazard ratio (95% CI), P, adjusted model 1</th>
<th>All-cause mortality, hazard ratio (95% CI), P, adjusted model 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>CSA</td>
<td>2.3 (1.4–4.0), 0.002</td>
<td>1.7 (0.9–3.3), 0.078</td>
</tr>
<tr>
<td>CVD</td>
<td>1.5 (0.9–2.6), 0.13</td>
<td>1.1 (0.6–2.0), 0.71</td>
</tr>
<tr>
<td>Diabetes</td>
<td>2.1 (1.3–3.6), 0.004</td>
<td>2.3 (1.4–3.9), 0.002</td>
</tr>
<tr>
<td>NTpro-BNP</td>
<td>3.8 (2.2–6.7), &lt;0.001</td>
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</tbody>
</table>

CSA, central sleep apnoea; CVD, cardiovascular disease; NT-pro-BNP, N-terminal fragment brain natriuretic peptide.

**Table 3.** Cox-regression models analysing the association between participants with CSA and cardiovascular mortality during 7-year follow-up

<table>
<thead>
<tr>
<th></th>
<th>CV mortality, hazard ratio (95% CI), P, adjusted model 1</th>
<th>CV mortality, hazard ratio (95% CI), P, adjusted model 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>CSA</td>
<td>2.3 (1.2–4.8), 0.018</td>
<td>1.5 (0.7–3.2), 0.32</td>
</tr>
<tr>
<td>CVD</td>
<td>2.1 (1.1–4.3), 0.037</td>
<td>1.6 (0.7–3.3), 0.24</td>
</tr>
<tr>
<td>Diabetes</td>
<td>2.9 (1.5–6.0), 0.002</td>
<td>3.1 (1.6–6.2), 0.001</td>
</tr>
<tr>
<td>NTpro-BNP</td>
<td>3.9 (1.9–8.3), &lt;0.001</td>
<td></td>
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</table>

CSA, central sleep apnoea; CVD, cardiovascular disease; NT-pro-BNP, N-terminal fragment brain natriuretic peptide.

SDB. NT-pro-BNP was significantly higher in those with CSA compared with those with OSA (P = 0.001) and to those with no SDB (P < 0.001). History of IHD (43%) and TIA stroke (24%) was significantly more common in those with CSA compared with those with OSA (IHD 23% and TIA/stroke 7%, P = 0.007, P = 0.002) and no SDB (IHD 21% and TIA/stroke 5%, P = 0.002, P < 0.001).

**Association of OSA and CSA to mortality**

During the mean of 1,911 follow-up days, the Kaplan–Meier analysis shows that there were significant associations between CSA and all-cause mortality (P = 0.006) and CV mortality (P = 0.046) (Figure 1A and B). Pairwise Log-rank comparisons showed that all-cause mortality and CV mortality were significantly higher for those with CSA compared with those with no SDB (35 versus 16%, P = 0.006 and 20 versus 9%, P = 0.039) and OSA (35 versus 15%, P = 0.005 and 20 versus 8%, P = 0.023). Participants with OSA did not significantly differ compared with those with no SDB concerning all-cause mortality (15 versus 16%, P = 0.82) or CV mortality (8 versus 9%, P = 0.80).

To examine whether CSA independently increased the risk for all-cause or CV mortality, a Cox-regression hazard analysis was performed with CVD, diabetes and
NT-pro-BNP as covariates. These covariates were entered in two blocks, first CVD and diabetes and lastly NT-pro-BNP. In the unadjusted analyses, CSA significantly increased the risk for all-cause ($P = 0.002$) and CV mortality ($P = 0.018$) by more than two times (Tables 2 and 3). After adding CVD and diabetes to the models, CSA remained as a significant predictor for all-cause mortality (HR: $1.7, P = 0.027$) but not for CV mortality (HR: $1.7, P = 0.17$). The association between CSA and all-cause mortality lost its significance after addition of NT-pro-BNP to the model.

**Discussion**

To our knowledge, no studies have focused on characteristics associated with OSA and CSA in community-dwelling elderly. We found that 55% had SDB at baseline. A potential bias for this high rate of SDB could be that a higher number of those who choose to participate in the sleep study also had more sleeping problems. However, our rate of SDB is comparable with the large-scaled Sleep Heart Health Study who reported 54% of SDB in persons aged 70–79 years [17]. The specific prevalence of OSA and CSA was not reported in that study. We found that 38% had OSA and 17% had CSA (Supplementary data are available in *Age and Ageing* online, Figure S1).

Overall, no great differences were found in the present study regarding demographical and clinical variables between participants with CSA, OSA or no SDB. However, those with CSA had, compared with those with no SDB and OSA, more impaired systolic function and CVD. It has been suggested that SDB in elderly and in younger people are two distinct conditions [5, 17]. In contrast to middle-aged people, SDB has in elderly been found to be weakly associated with BMI or hypertension [17, 18]. This is in line with our study, where BMI, MAP and hypertension were associated neither to OSA nor CSA [19, 20]. In middle-aged people, OSA is associated with the development of CV morbidity and mortality [9]. A potential explanation for this is that OSA, because of the frequent cycles of desaturations followed by rapid re-oxygenation, can cause oxidative stress, inflammation and sympathetic activation [19, 20]. This may promote atherosclerosis [19, 20], and impairment of systolic function [21]. Hence, OSA may therefore be considered as a potential cause for CVD, whereas CSA could be an effect of a CVD and impaired systolic function. In this study, performed in a population mean aged 78 years, OSA was, however, not associated with CVD or mortality. One mechanistic explanation for this may be that the AHI and nadir saturation during apnoeas and hypopnoeas becomes less severe with a higher age [5]. Another possible explanation could be the ‘survival of the fittest’, i.e. that OSA causes more CV deaths earlier in life. In patients with HF, who generally are older, prevalence rates for CSA ranges between 15 and 40% [22, 23] and, CSA have been found to be associated with high plasma levels of BNPs [24, 25]. The presence of CSA in our population may, therefore, reflect the severity of CVD and cardiac function.

We found that CSA, but not OSA, increased the risk for all-cause and CV mortality, but the association of CSA was not, after adjustments for several covariates, independent neither for all-cause nor CV mortality. In other studies [26–28] performed on elderly people, SDB has not been associated with mortality. However, none of these have, as we did, analysed OSA and CSA specifically. In patients with HF, CSA has independently been associated with mortality [12]. In the model for all-cause mortality, we found that the association of CSA was independent after adjusting for CVD and diabetes, and there was a trend to significance after adjustment for NT-pro-BNP (HR: $1.5, P = 0.078$). This implies that the presence of CSA may accelerate the progression of CVD and the impairment of cardiac function. However, our CSA population was small ($n = 54$) and the findings should therefore be interpreted cautiously. Different approaches for treatment of CSA in patients with HF have been evaluated [2]. There are indications that suppression of CSA by continuous positive airway pressure might improve cardiac function and survival in middle-aged patients with HF [29]. More interventional studies are obviously needed, especially in people with higher age.

**Conclusion**

More than half (55%) of community-dwelling elderly in this sample had SDB. OSA was found in more than one-third (38%), whereas almost one-fifths (17%) had CSA. OSA did not differ concerning demographic and clinical characteristics and prognosis compared with those with no SDB. In contrast, CSA was associated with CVD and impaired systolic function and higher mortality. However, the association to mortality was not independent after adjusting for CVD, diabetes and NT-pro-BNP. The findings in this study suggest that OSA in persons >75 years may be seen as less important, whereas CSA might be a pathological marker of CVD and impaired systolic function associated with higher mortality. More studies are needed to examine whether CSA is an independent factor that impairs the prognosis for elderly people.

**Key points**

• A total of 55% of community-dwelling elderly had SDB. OSA was found in 38% and CSA in 17%.
• Community-dwelling elderly with OSA compared with those without SDB are not obviously different regarding clinical characteristics and prognosis.
• Community-dwelling elderly with CSA have more of CV diseases and impaired systolic function but CSA is not independently associated with mortality.
• More studies are needed to establish whether CSA among community-dwelling elderly is a factor that independently is associated with mortality.
Conflicts of interest
None declared.

Funding
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Supplementary data
Supplementary data mentioned in the text is available to subscribers in Age and Ageing online.

References
Older people’s preferences regarding programme formats for managing concerns about falls

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Abstract

Objective: to explore the preferences of community-dwelling older persons regarding different programme formats for managing concerns about falls.

Subjects and design: cross-sectional study of 5,755 community-dwelling people aged ≥70 years in the Netherlands.

Methods: a questionnaire assessed people’s willingness to participate per programme format (n = 6), i.e. a programme at home, via telephone, via home visits and telephone consultations, via television or via Internet.

Results: of the 2,498 responders, 62.7% indicated no interest in any of the formats. The willingness to participate per programme format varied between 21.5% (at home) and 9.4% (via Internet). Among people interested in at least one of the formats (n = 931), higher levels of fall-related concerns were associated with increased preference for a programme with home visits. Poor perceived health and age ≥80 years were associated with less preference for a group programme. Higher educated people were more in favour of a programme via Internet compared with their lower educated counterparts.

Conclusion: the majority of community-dwelling older people are not likely to participate in any of the six proposed programme formats for managing concerns about falls. However, when diverse formats of effective programmes will be made available, uptake and adherence may be increased since programme preferences are associated to specific population characteristics.

Keywords: aged, fear of falling, programme formats, patient preference, accidental falls, elderly