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Evaluation of academic detailing visits on GP knowledge and practice for statin use and management

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File S1.

NPS MEDICINEWISE PROFESSIONAL

STATINS: OPTIMISING THERAPY, ADDRESSING INTOLERANCE

Assess absolute cardiovascular risk before prescribing lipid-modifying medicines

- Interpreting lipids in the context of absolute cardiovascular (CV) risk, rather than as an isolated risk factor, remains the most comprehensive and effective approach to lipid management.[1,2]
- Baseline absolute CV risk, as well as extent of LDL-C reduction, are key factors in determining CV outcomes in patients on statin therapy.[3]

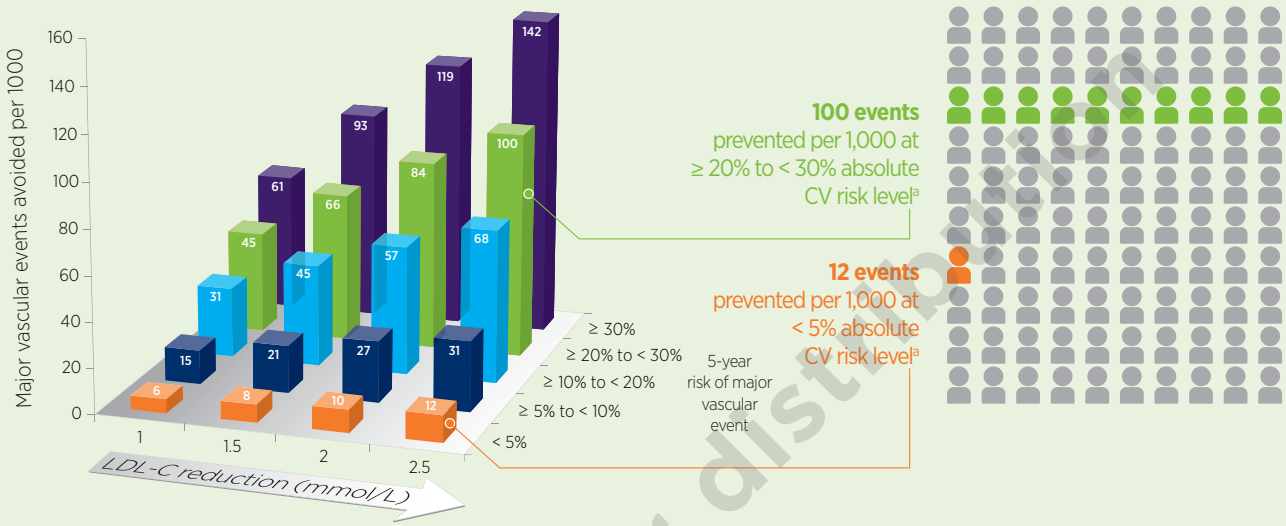

FIGURE 1 Predicted number of 5-year major vascular events prevented per 1,000 with LDL-C reductions from statin therapy at different absolute CV risk levels[3]

Bar graph adapted with permission from Cholesterol Treatment Trialists and Collaborators. Lancet 2012;380:581–90.
[a] 2.5 mmol/L reduction of LDL-C with statin therapy

- Guidelines remain clear on an absolute risk approach to guide treatment, strongly recommending:[1,2]
  - **High absolute CV risk or established CVD**
    - ➔ Prescribe lipid-modifying medicines with lifestyle modification
  - **Moderate absolute CV risk**
    - ➔ Try lifestyle modification before considering lipid-modifying medicines
  - **Low absolute CV risk**
    - ➔ Encourage lifestyle modification; recognise that lipid-modifying medicines are usually not required
- Involve your patients in decision making by:[4,5]
  - explaining the concept of absolute CV risk
  - counselling them about their risk score and cholesterol level
  - offering information about the absolute benefits and harms of treatment options

Choosing Wisely Australia

RACGP Choosing Wisely recommendation states:[6] Don't commence therapy for hypertension or hyperlipidaemia without first assessing the absolute risk of a cardiovascular event.

Have your low CV risk patients been unnecessarily prescribed statins?

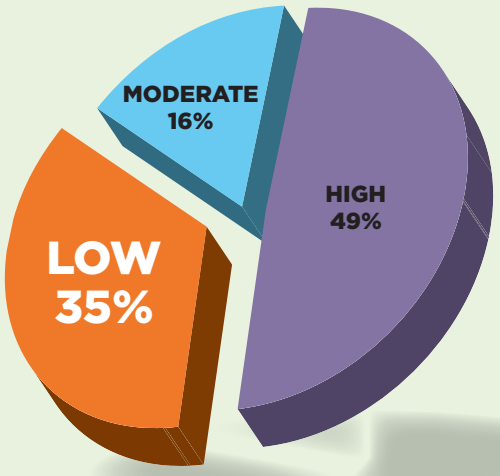

FIGURE 2 Proportion of patients[b] currently prescribed statins who were at low, moderate and high absolute CV risk before starting treatment.

[b] Australian general practice: MedicineInsight data: n=53,442[7]

Optimise LDL-lowering by adequately trialling statin therapy before adding a second agent[4]

START STATIN

- **Check baseline** CK, ALT and blood glucose levels.[8]
- **Set targets**[1]

TC	< 4.0 mmol/L
HDL-C	≥1.0 mmol/L
LDL-C	< 2.0 mmol/L primary prevention
	< 1.8 mmol/L secondary prevention[c]
Non-HDL-C	< 2.5 mmol/L
TG	< 2.0 mmol/L

Manage secondary prevention more aggressively than primary prevention. Set lower LDL-C targets and consider initiating statin therapy at a higher intensity (dose + potency).[4,9,10]

- **Counsel patients** about what to expect when taking statins.[11]

[c] Target recommended for patients with acute coronary syndrome (ACS) and coronary heart disease based on Australian guidelines,[9,10] and also applied to patients with a history of stroke, transient ischaemic attacks and peripheral vascular disease, as per common practice and European guidelines.[12]

CHECK AT 4–8 WEEKS

Lipid profile

- Maximum response expected within 4 weeks of starting therapy or increasing dose.[8] However, individual response is variable (due to non-adherence, lifestyle, biological and, possibly genetic factors), so dose titration may be required.[12,13]

Non-fasting blood samples make no significant difference and may improve patient adherence compared to fasting samples, according to 2016 European guidelines.[14] Non-fasting samples are increasingly accepted in Australia.[15] Fasting is still required in some cases, such as when triglycerides are elevated.[14]

Adherence

- Non-adherence is a top predictor of failure to meet targets.[13] Non-adherence rates up to 67% after 12 months have been found.[16] Effective methods for improving adherence include providing education, simplifying drug regimens, pharmacist review and reminders (eg, MedicineList+).[1]

Lifestyle

- Provide advice, support and pharmacotherapy where appropriate for stopping smoking, improving diet and increasing physical activity, such as the approach recommended in The Redbook.[1,2]

Adverse effects

- Check CK and ALT 4–8 weeks after initiation and dosage adjustment.[8] To avoid detection of asymptomatic CK or ALT elevation, routine blood tests are not needed unless clinically indicated.[8]
- If muscle symptoms develop, see SAMS Assessment Guide and SAMS Management Algorithm (insert).
- Check blood glucose for impaired glucose metabolism.[8]

MEETING TARGETS

For most patients statin therapy is lifelong.

Primary prevention

- Measure lipids every 6–12 months during maintenance.[17]
- Consider reducing or withdrawing statins for those who make significant and sustained lifestyle changes that reduce absolute CV risk to low (eg, stopping smoking, losing 10% to 20% body weight). Monitor for at least 12 months afterwards to ensure a sustainable positive impact.[1]

Secondary prevention

- Continue aggressive approach to management.[10]

NOT MEETING TARGETS

Adequately trial statin therapy

- Titrate statin to maximum tolerated dose (MTD).[1,8] Dose increases are made at intervals[17] of 4–8 weeks.[8]
- Check adherence to medicines and lifestyle changes.

If patient persistently not meeting targets

- Consider adding a second agent – ezetimibe, bile acid-binding resin, nicotinic acid, fibrates or fish oil. Choice depends on lipid profile, target goals and patient factors.[1,4,8,17]

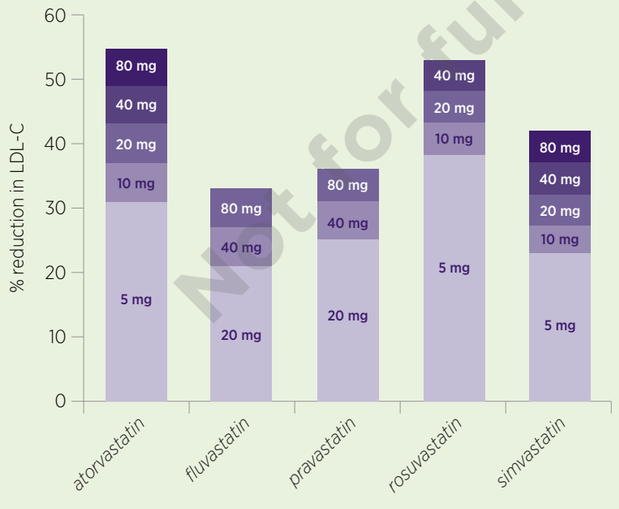

FIGURE 3 Effect of statins on LDL-C according to dose[18]

Adapted with permission from Law MR, et al. BMJ 2003;326:1423–7

TABLE 1 **Examples of cytochrome P450-mediated statin medicine interactions**[17]

STATIN	METABOLISED BY	STATIN CONCENTRATION MAY BE INCREASED BY	STATIN CONCENTRATION MAY BE DECREASED BY
Atorvastatin **Simvastatin**	CYP3A4 (main)	CYP3A4 inhibitors • Azole antifungals (all) • Calcium channel blockers (only diltiazem, verapamil) • Fluvoxamine • Grapefruit juice • HIV-protease inhibitor antiretrovirals (all) • Macrolide antibacterials (only clarithromycin, erythromycin) • Ticagrelor	CYP3A4 inducers • Antiepileptics (some eg, carbamazepine, phenytoin) • HIV-protease inhibitor antiretrovirals (only ritonavir, tipranavir) • Rifampicin • St John's wort
Fluvastatin	CYP2C9 (main) CYP3A4 (lesser extent)	CYP2C9 inhibitors • Amiodarone • Azole antifungals (only fluconazole, voriconazole) • SSRIs (only fluoxetine, fluvoxamine) CYP3A4 inhibitors (see above)	CYP2C9 inducers • Rifampicin • St John's wort CYP3A4 inducers (see above)
Pravastatin **Rosuvastatin**	Not significantly metabolised by CYP enzymes		

Ezetimibe

- Patients not meeting LDL-C targets on optimised statin therapy may benefit from the addition of ezetimibe[1,8] which can achieve a further 15% to 23% reduction in LDL-C.[19]
- Limited data is available for CV outcomes for ezetimibe and statin combination. In the IMPROVE-IT trial involving post-ACS patients, compared to simvastatin alone, combination with ezetimibe further reduced the risk of CV events.[20]

Fibrates and fish oil for triglycerides

- Fibrates and/or fish oil are indicated for predominant TG elevation, particularly if HDL-C is low or TG level is > 10 mmol/L. Fibrates are also used with statins for the management of mixed hyperlipidaemia (elevated TG and LDL-C).[8]

PCSK9 inhibitors

- Proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitors are a new class of injectable, monoclonal antibody lipid-modifying agents. They are reserved for heterozygous familial hypercholesterolaemia (FH)[21,22] and only evolocumab is PBS-listed for homozygous FH.[23]
- PCSK9 inhibitors reduced LDL-C by 57% to 61% in phase 2 and 3 trials.[24] FOURIER trial (evolocumab) demonstrated a positive composite primary CV endpoint (median follow-up 2.2 years).[25] Overall, safety and efficacy data are currently limited by a lack of longer term studies.[25,26]

NPS MEDICINEWISE STATINS: OPTIMISING THERAPY, ADDRESSING INTOLERANCE

TABLE 2

Classes of single active ingredient lipid-modifying medicines

Based on Australian Medicines Handbook (AMH) unless otherwise stated; check product information for full details.[17]

CLASS active ingredients	LDL-C LOWERING (monotherapy compared to placebo)[d]	ADVERSE EFFECTS (> 1%)	CONSIDERATIONS
Statins • atorvastatin • fluvastatin • pravastatin • rosuvastatin • simvastatin	21% to 55%[18] (range varies according to dose; see Figure 3)	Myalgia, mild transient GI symptoms, headache, sleep disturbance (eg, insomnia, nightmares), dizziness, elevated aminotransferase concentrations. Rosuvastatin 40 mg: proteinuria usually transient and not associated with worsening renal function.	**Contraindication:** pregnancy,[b] concurrent sodium fusidate use. Simvastatin: concurrent use with some CYP3A4 inhibitors, gemfibrozil, cyclosporin or danazol. Rosuvastatin 40 mg: Asian ancestry. **Precautions:** severe intercurrent illness (infection, metabolic disorder), myopathy with other lipid-modifying medicine, renal and hepatic impairment. **Medicine interactions:** CYP450 interactions (see Table 1), cyclosporin, other medicines that cause myopathy eg, nicotinic acid, colchinine. **Dosing time:** pravastatin and simvastatin may be slightly more effective taken in the evening, but irrelevant if adherence is compromised.
Ezetimibe	18% to 20%[27]	Headache, diarrhoea.	**Precautions:** concurrent fenofibrate use, moderate-severe hepatic impairment.
Bile acid-binding resins • cholestyramine • colestipol	18% to 25%[12]	Constipation, abdominal pain, dyspepsia, flatulence, nausea, vomiting, diarrhoea, anorexia. Adverse effects are dose-related; minimise by starting with low dose and increasing gradually.	**Precautions:** TG > 3 mmol/L, complete biliary obstruction, constipation, diverticular disease, severe haemorrhoids. Cholestyramine: PKU. **Vitamin supplementation:** consider fat-soluble vitamin supplements for high doses over extended period. **Timing:** can reduce effect of other medicines; take other medicines at least 1 hour before or 4–6 hours after.
Fibrates • fenofibrate • gemfibrozil	5% to 15%[17]	GI disturbances (eg, dyspepsia, abdominal pain), increased CK concentration (reversible). Myopathy (concurrent statin use; fenofibrate less risk than gemfibrozil). Gemfibrozil: headache, dry mouth, myalgia. Fenofibrate: increased aminotransferase concentration.	**Contraindications:** severe renal or hepatic impairment, primary biliary cirrhosis, gallstones, gall bladder disease, photosensitivity due to a fibrate. Gemfibrozil: concurrent simvastatin or dasabuvir use. Fenofibrate: pancreatitis unless due to hypertriglyceridaemia, concurrent ketoprofen use. **Precautions:** fenofibrate: concurrent ezetimibe or thiazolidinedione use. **Sun exposure:** avoid skin exposure (use protective clothing, sunscreen). **Biochemistry:** complete blood count and liver function at baseline and during treatment; CK at baseline, repeat if clinically indicated.
Nicotinic acid (niacin)	15% to 18%[12]	Vasodilation, hypotension, dyspepsia, diarrhoea, nausea, vomiting, hyperpigmentation, and face and neck flushing.	**Contraindications:** pregnancy,[e] symptomatic hypotension, recent MI (seek specialist advice). **Precautions:** peptic ulcer disease, gout, diabetes, coronary artery disease, CrCl < 30 mL/minute, history of jaundice or hepatic disease, treatment with antihypertensives.
PCSK9 (proprotein convertase subtilisin/ kexin type 9) inhibitors • alirocumab • evolocumab	57% to 61%[24]	Injection site reactions (mild pain, redness), nasopharyngitis, upper respiratory tract infections, influenza, pruritus.[17,22]	**Precautions:** allergic reactions, immunogenicity.[21,22] Alirocumab: severe hepatic impairment.[22] **Administration:** fortnightly or monthly subcutaneous injection.[21,22]
Fish oils (omega-3 fatty acids) • docosahexa-enoic acid (DHA) and eicosapenta-enoic acid (EPA).[28]	No change[17]	Mild GI effects (belching, nausea, diarrhoea, fishy taste).[f,28]	**Precautions:** concurrent anticoagulant use,[12] high doses may increase bleeding time.[17] **Dosage:** 2–4 g daily omega-3 fatty acids to lower triglycerides.

[d] These medicines have effects on other lipids, which are not reported here. [e] Statins and nicotinic acid contraindicated in pregnancy; for other information on pregnancy and breastfeeding for all classes, see product information. [f] Most common adverse effects (not defined by %)

References available online at: **nps.org.au/statins-card-refs**

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May 2017 NPS1781

File S2.

NPS MEDICINEWISE PROFESSIONAL

Statin-associated muscle symptoms (SAMS)

Use a systematic approach to assess suspected statin intolerance

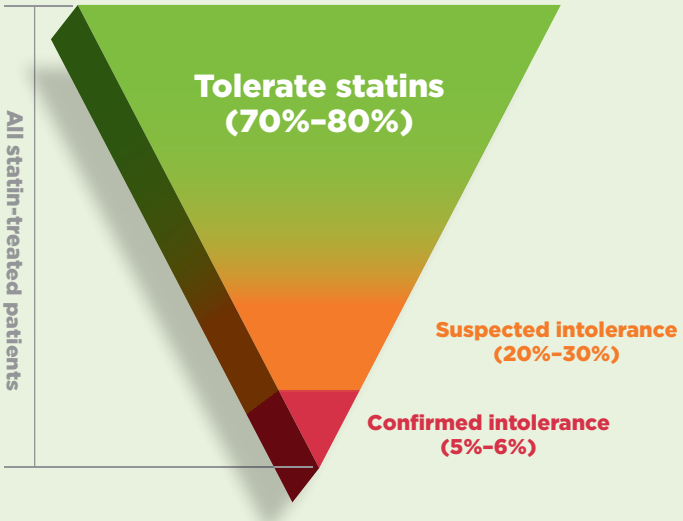


FIGURE A **Tolerability of statin treatment**[1]

- Statin intolerance is rarely life-threatening and may have a lower incidence than is commonly reported.[2–4]
- Statins have been associated with a nocebo effect, whereby patients experience adverse effects based on the expectation of harm from a treatment.[5]
- For muscle-related adverse effects:
  - Incidence of statin-associated myalgia is lower in blinded RCTs (1% to 5%)[6] compared to observational studies (7% to 29%).[4]
  - Myopathy incidence is ~ 1 in 10,000 per year.[4]
  - Rhabdomyolysis incidence is ~ 1 in 100,000 per year.[4]
- Involve patients in assessing and managing adverse effects.
- Advise patients to contact you if they experience muscle symptoms, and not to stop taking their statin.[6]

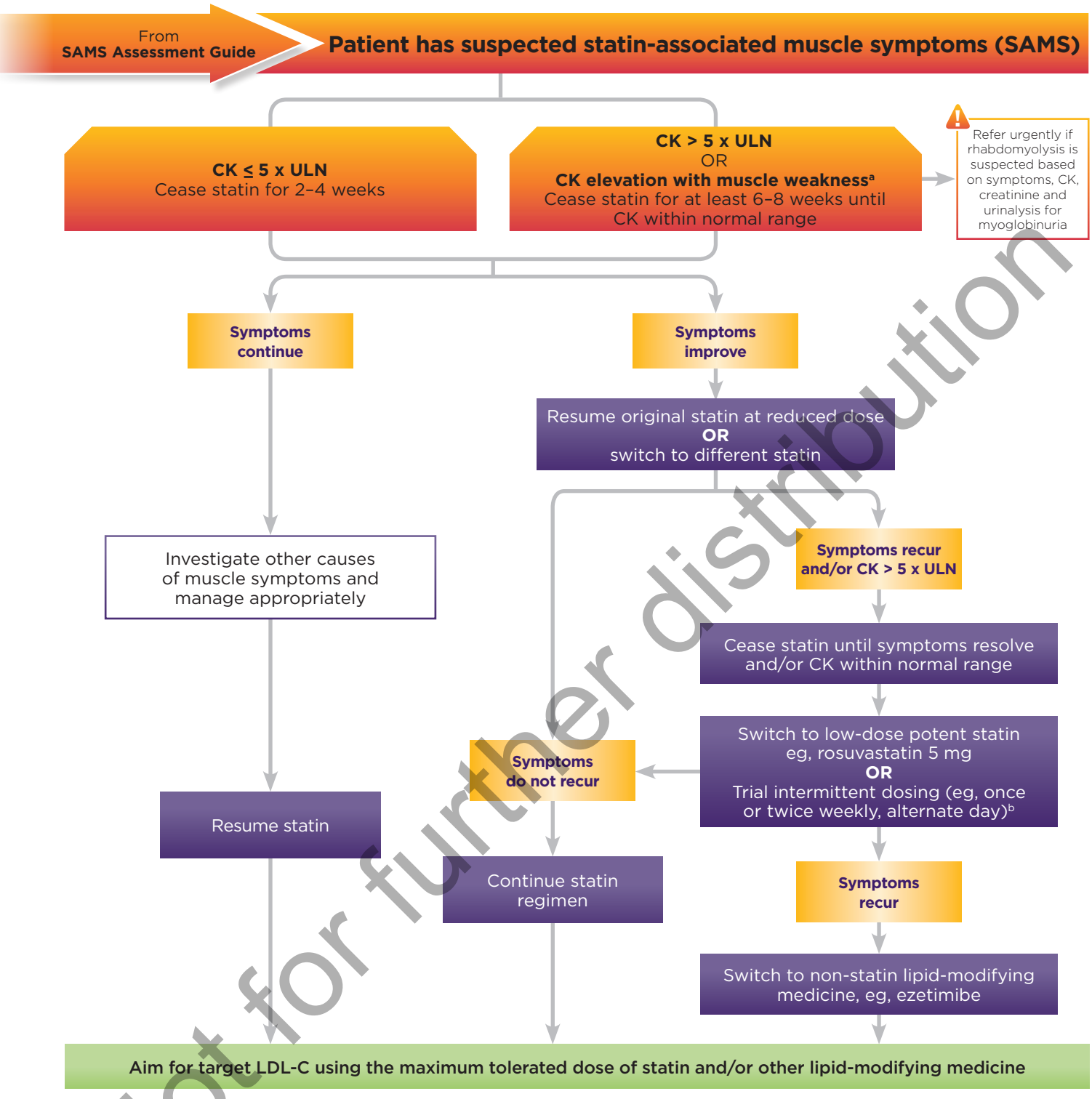
SAMS Assessment Guide

SAMS LESS LIKELY		SAMS MORE LIKELY
Unilateral Non-specific distribution Tingling, twitching, shooting pain, nocturnal cramps or joint pain	**Nature of symptoms**[4,6,7]	Bilateral Large muscle groups (eg, thighs, buttocks, calves, shoulder girdle) Muscle ache, weakness, soreness, stiffness, cramping, tenderness or general fatigue
Onset before statin initiation Onset > 12 weeks after statin initiation	**Timing of symptoms**[4]	Onset 4–6 weeks after statin initiation Onset after statin dosage increase
Non-statin causes of muscle symptoms including: • conditions eg, hypothyroidism, polymyalgia rheumatica • vitamin D deficiency • unaccustomed/heavy physical activity • medicines eg, glucocorticoids, antipsychotics, immunosuppressant or antiviral agents	**Other considerations**[4,7]	Risk factors for SAMS including: • medicine or food interactions • high-dose statin therapy • history of myopathy with other lipid-modifying medicines • regular vigorous physical activity • impaired hepatic or renal function • substance abuse (eg, alcohol, opioids, cocaine) • female • low BMI
	CK levels[4]	Elevated (> ULN; but may also be normal) Elevated CK levels decrease after statin ceased

If SAMS is likely, proceed to the **SAMS Management Algorithm (see overleaf)**

References available online at:
nps.org.au/statins-card-refs

SAMS Management Algorithm



CK = creatine kinase, LDL-C = low density lipoprotein cholesterol, ULN = upper limit of normal

[a] CK > ULN and weakness demonstrated upon physical examination. [b] Higher potency statins with a long half-life are preferred for intermittent dosing eg, rosuvastatin and atorvastatin

Acknowledgements

Developed based on the 2012 Therapeutic Guidelines: Cardiovascular and 2016 European Society of Cardiology/European Atherosclerosis Society Guidelines for the management of dyslipidaemias, with input from experts:

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File S3.

NPS MEDICINEWISE

Feb 2018

Statins: Optimising therapy, addressing intolerance

You recently participated in an educational activity on the topic *Statins: Optimising therapy, addressing intolerance* with an NPS MedicineWise Clinical Services Specialist. Your responses to this survey will help us assess this program and provide us with information to better support GPs.

A number of questions ask you to provide an answer for two different time periods. The first period (NOW) refers to your current attitudes and practice. The second period (BEFORE) refers to your attitudes and practice before participating in the program.

We appreciate your time and assistance. Your responses are confidential and will be reported in an aggregated, **de-identified** format. Please return your completed questionnaire by **3rd April 2018** using the enclosed reply-paid envelope, or to the address below.

Please mark your answers by crossing the box as instructed in the questions. ☒

NPS MedicineWise
Reply Paid 1980, Strawberry Hills, NSW 2012

1. Please place a cross in the box that best indicates your position on the following statements NOW and BEFORE participating in the program.

	NOW					BEFORE				
	Strongly Agree	Agree	Neutral	Disagree	Strongly Disagree	Strongly Agree	Agree	Neutral	Disagree	Strongly Disagree
Using the absolute CV risk enables the most effective approach to lipid management.	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Adherence to statin medicines should be checked at each consultation.	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Addition of a second lipid modifying medicine should be reserved for patients who have adequately trialled statin therapy.	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
In secondary prevention (patients with established CVD), an intensive approach to LDL-C lowering is usually warranted.	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
There is evidence to support a continuous, graded relationship between LDL-C and major CV events.	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Statins have a robust evidence base for efficacy and safety with over 30 years of clinical trial data.	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Ezetimibe has a strong evidence base for improving CV outcomes in both the primary and secondary prevention setting.	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Up to 90% of patients who cannot tolerate a statin will be able to tolerate an alternate statin.	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

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2. **When initiating a patient on statin therapy, please put a cross in the box that best indicates how frequently you would do the following NOW and BEFORE participating in the program.**

	NOW					BEFORE				
	Always	Often	Sometimes	Rarely	Never	Always	Often	Sometimes	Rarely	Never
Check baseline CK.	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Check blood glucose levels at baseline and at 4-8 weeks after initiating the statin.	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Counsel patients on what to expect when taking statins.	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Order ongoing ALT and CK tests every 3 months after initiating statins.	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Check for drug interactions with the statin.	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

3. **When considering prescribing a lipid-modifying medicine, which of the following strategies do you use to estimate CV risk in patients aged 45 – 74 years? Please indicate what you do NOW and what you did BEFORE participating in the program.** (Select all that apply. If not applicable leave blank)

	NOW	BEFORE
Review a patient's individual BP, lipid and glucose blood results.	☐	☐
The online Australian absolute CV risk calculator (cvdcheck.org.au).	☐	☐
The Heart Foundation Australian CV risk charts.	☐	☐
In-built clinical software CV risk calculators.	☐	☐
I don't generally estimate CV risk.	☐	☐
Other (please specify)_____	☐	☐

4. **Mandy is a 58-year-old patient at high absolute CV risk of 27% in the next 5 years, with diabetes, dyslipidaemia and a 30 pack/year history of smoking. You advised Mandy to quit smoking, provided appropriate resources and suggested she improve her lifestyle. She agreed to start Atorvastatin 20mg daily. 12 weeks later you order non-fasting lipid tests and her LDL-C is still not at target (LDL-C 2.7 mmol/L, ~40% reduction from baseline). How would you address this NOW and BEFORE participating in the program?** (Select all that apply. If not applicable leave blank)

	NOW	BEFORE
Check that Mandy is regularly adherent to her Atorvastatin.	☐	☐
Re-order fasting lipids as this is more accurate and would likely change your management.	☐	☐
Check how well Mandy has improved her lifestyle (exercise and diet).	☐	☐
Titrate her Atorvastatin dose up to 40mg daily and check how she tolerates it at the next consultation.	☐	☐
Add a second agent to help Mandy meet her target (e.g. ezetimibe).	☐	☐

5. **Max is a 55-year-old patient who comes in for a regular check-up. He has a history of GORD and IBS but does not take any regular medicines. He has a BP of 128/85 mmHg and BMI of 29 kg/m2. His lipid results are – TC 6.0 mmol/L, LDL-C 3.9 mmol/L, HDL-C 0.9 mmol/L, TG 2.8 mmol/L. What is your first step to address Max's lipid profile? Please indicate what you would do NOW and what you would have done BEFORE participating in the program**. (Select ONE response only for NOW and ONE response only for BEFORE)

	NOW	BEFORE
Initiate low dose statin therapy as this is first line therapy for predominant elevation of LDL-C.	☐	☐
Advise Max to intensively change his diet and lifestyle which should reduce his lipids to target.	☐	☐
Initiate a high intensity statin (e.g. Rosuvastatin 40mg) in order to achieve the 50% reduction in LDL-C that he needs to meet targets.	☐	☐
Assess Max's absolute CV risk using the Australian CV risk calculator.	☐	☐

6. **Which of the following factors are MOST suggestive of statin associated muscle symptoms in patients taking a statin? Please indicate how you would respond NOW and BEFORE participating in the program.** (Select all that apply. If not applicable leave blank)

	NOW	BEFORE
Nocturnal muscle cramps.	☐	☐
Muscle ache, weakness, soreness, stiffness or general muscle fatigue.	☐	☐
Muscle symptoms in a female patient with low BMI.	☐	☐
Muscle symptoms in patients with severe Vitamin D deficiency.	☐	☐
Muscle symptoms with elevated CK (above the upper limit of normal, ULN) which normalises after cessation of the statin.	☐	☐

7. **How would you manage a patient on a statin with muscle soreness and a CK level of 3 x ULN? Please indicate what you would do NOW and what you would have done BEFORE participating in the program.** (Select all that apply. If not applicable leave blank)

	NOW	BEFORE
Cease the statin for 2-4 weeks and monitor for improvement of muscle symptoms.	☐	☐
Switch to a non-statin lipid-modifying medicine like ezetimibe.	☐	☐
If symptoms improve after discontinuation, reduce the statin dose or switch to an alternate statin.	☐	☐
Refer the patient to hospital as they may have rhabdomyolysis.	☐	☐

About your participation in NPS MedicineWise activities

8. **In December 2017, you may have received your Department of Human Services confidential prescribing data related to lipid-modifying medicines.**

Did the prescribing feedback?	Yes	No	Can't recall/ Didn't receive
Present your prescribing data in a way that was easy to understand	☐	☐	☐
Provide a useful tool for comparing your prescribing activity to your peers	☐	☐	☐
Help you to reflect on your prescribing of lipid-modifying medicines	☐	☐	☐
Prompt a change in your prescribing of lipid-modifying medicines	☐	☐	☐

9. **Which NPS MedicineWise activities in the *Statins: Optimising therapy, addressing intolerance* program did you participate in** (Select all that apply):

- ☐ One-to-one educational visit
- ☐ Small-group meeting
- ☐ Clinical e-Audit: *Statins: who, when and how?*
- ☐ Online case study: *Optimising statin therapy*
- ☐ Read the Medicinewise News: *Uncovering the truth about statin intolerance* (available on the NPS MedicineWise website)
- ☐ Do not recall

10. **Have you participated in any other (non-NPS MedicineWise) educational activities about statins in the last 12 months?**

- ☐ No ☐ Can't recall
- ☐ Yes (please specify):_____

11. **Have you used any of the following NPS MedicineWise patient resources in discussions with your patients (available on the NPS MedicineWise website)?** Select ALL that apply.

- ☐ Statins Patient Action Plan for assessing and managing muscle symptoms
- ☐ Statin medicines FAQs
- ☐ I am not aware of these resources

About you and your practice

12. **Are you?**

☐ Male ☐ Female

13. **How many years have you practised as a GP?**

|_|_|

14. **Approximately how many patients would you see in a normal week?**

|_|_|_|

15. **Approximately how many patients would you see for a statin-related visit in a normal week?**

|_|_|_|

16. **What is the postcode of your main place of work?**

|_|_|_|_|

17. **Your principal practice has?**

- ☐ 1 GP — solo practice
- ☐ 2 GPs
- ☐ 3–5 GPs
- ☐ 6–8 GPs
- ☐ More than 8 GPs

Thank you for completing this questionnaire.

Your responses are highly valued. If you have any queries please contact Dr Isla Hains, NPS MedicineWise Program Evaluation on (02) 8217 9235