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Fracture prevention by stimulating physical activity via a mass media campaign

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1.1 Definition of intervention

The intervention is a media campaign to promote regular moderate-intensity physical activity (PA) targeting adults aged 25 to 60. The campaign makes use of television and print-media advertising, physician mail-outs and community-level support programs and strategies. It is modelled to resemble a campaign conducted in NSW in 1998 [1].

1.2 Health states / risk factors affected by the intervention

This analysis is limited to the reduction in osteoporosis-mediated fractures of the hip, spine (vertebrae), pelvis, clavicula/humerus (shoulder/upper arm), rib, wrist, hand, lower leg and foot. The effects are mediated by an increase in bone strength and improved muscle strength and coordination, and add to benefits in reducing cardiovascular disease, diabetes and cancer presented in an earlier ACE Prevention paper [2]. This paper presents the effect of adding fractures as an outcome to the results presented in this earlier paper. The results for fractures are presented separately to enable comparison with other interventions that aim to reduce (osteoporotic) fractures.

1.3 Current Practice

This analysis assumes that the current fracture rates are influenced by the current levels of physical activity. The intervention effect is considered to add to any effect current drug treatment has on fracture rates. Recently, about 7% of women and 2% of men aged 59 years and over received pharmaceutical treatment for osteoporosis [3].

1.4 Efficacy / Effectiveness of the intervention

The campaign led to an increase of 0.64 hours of physical activity per week in NSW compared to other states in the targeted 25 to 60 year age group [1]. (Effects at other ages were not assessed.)

To model the effects of this intervention on fracture risks, ideally our model would be based on the results of randomised controlled trials (RCTs) with physical activity as the exposure category and fracture risk as the outcome. However, such studies have not been done – because fractures are infrequent, they would require enormous sample sizes. We therefore

model two pathways to estimate the effect on fracture rates: via bone mineral density (BMD) and via the frequency of falls. Both pathways make use of evidence collected in RCTs.

In the 'BMD-pathway' we translated the increase in physical activity into a change in the average bone mineral density by age. We based the BMD increase on a meta-analysis that shows that a wide range of different exercise-programmes result in an average BMD-increase of 0.89% at the femoral neck for every year of programme [4]. Given that the programmes added an average of 3 hours of PA per week, one hour of PA per week during a year adds 0.3% to the average BMD and the campaign results in a 0.19% increase in BMD. The spread in BMD by age and sex were kept constant; the entire distribution shifted upward as the average BMD increased. Consistent with the earlier analysis of the impact of physical activity on CVD, DM and cancer risks, we assumed that the PA-effect of a one-off campaign declines at 50% per year. Over the years this results in the equivalent of two years of physical activity at the above level of intensity, which leads to an estimated average 0.38% increase BMD. This leaves the question of how long the extra BMD can be assumed to remain present. The evidence is limited (see appendix 3) but suggests that physical activity among young adults results in permanent gains in BMD, though the difference with less active persons does decline over time. For postmenopausal women, the evidence suggests that the extra BMD is quickly lost after physical activity levels return to their pre-intervention levels. In our base case analysis we take the cautious assumption the extra BMD is lost at the same rate as physical activity (50% per year). In a sensitivity analysis we also explore the most optimistic assumption – that the extra BMD gain by extra physical activity remains present for the remainder of life – and 3 intermediate scenarios.

For the 'falls-pathway' we used a Cochrane meta-analysis that links physical activity levels to fall frequency [5] and assumed the reduction in falls would result in an equal reduction in fractures. In this review, an average of 2 hours of physical activity per week was added, which led to an 11% reduction in falls (RR = 0.89; 95% CI 0.78, 1.01). In the base case analysis we assume that the effect remains only as long as the extra PA is practiced (and then shows a decay of 50% per year). In a sensitivity analysis we include a scenario with a slower loss of the fall-prevention effect (decay of 25% per year).

The two pathways reflect different mechanisms: one via a strengthening of bone, the other via improved balance and muscle strength and a concomitant reduction in falls. In our model we multiplicatively combine the results of the two pathways. (For example, if both pathways decrease a risk by 10% each, the combined effect is a 19% risk reduction).

1.5 Modelling to health outcomes

The model mimics the 2003 Australian population and extrapolates to future years, assuming no trends in the incidence of fractures. It compares the incidence of fractures in scenarios with and without the media campaign.

For the BMD-pathway the model uses population distributions of BMD, relative risks for fracture by BMD and age, and the average change in BMD calculated as described above. The analysis via falls directly links physical activity to fracture frequency. The health impact of different fractures is estimated in a proportional multi-state life table.

The population distribution of BMD by age is fitted to data from the Geelong and Dubbo studies [6, 7]. The threshold for osteoporosis has been defined on the same data, using the WHO definition (a BMD that is lower than 2.5 standard deviations below the mean for 25-year old women). In combination with age- and BMD level-specific fracture relative risks [8] (Appendix 2), this allows calculation of the average fracture risk for women with osteoporosis. Physical activity increases BMD as described in section 1.4.

Incidence and mortality of fractures were estimated in the Australian Burden of Disease 2003 study ('Aus BoD'). Mortality was analysed separately for fractures of the hip (85% of the total number of osteoporosis-related deaths), pelvis, clavicle, rib and spine, and was assumed to occur within a year after fracture. In addition, survivors of a hip fracture were exposed to a 20% increased risk of death [9] (i.e., this concerns deaths that were not coded to fractures). Hip fracture cases were assumed to have a 29% chance of long-term disability with severity estimated at a disability weight level of 0.272 [10]. Half was attributed to osteoporosis and half to pre-existing frailty.

1.6 Costs of interventions and offsets

The costs for a national media campaign were estimated at $13.3 million by up scaling from the 1998 NSW campaign targeting adults aged 25 to 60 who were motivated but insufficiently active [1] and inflating to 2003 values (see [11]). Costs of an Australia-wide mass media intervention were derived from cost estimates for the NSW Health campaign in 1999. Key components include the cost of printing and developing materials (e.g. television commercial, brochures and posters), buying television media, community service announcements (delivered free of charge), supplementary grants for community development initiatives and tracking surveys [12]. All component costs were inflated to 2003 dollars using the Consumer Price Index (ABS). Costs were scaled up, where relevant, to a

magnitude associated with a six-week campaign targeting 9.74 million Australia-wide (rather than a four-week campaign targeting 1.4 million in New South Wales), then summed to determine the total cost of a mass media campaign intervention in Australia. No costs relating to the extra physical activity incurred by the population were included. Cost offsets in the first year post-fracture were based on the Dubbo study [13]. As opposed to the custom in ACE-Prevention, the health care costs in added years of life were included in the calculations for this intervention. This was done because excluding these costs would lead to the misleading result that adding an extra beneficial health effect (the reduction in fractures) would paradoxically increase future health care costs. This result would be an artefact arising from the inclusion in this model of costs due to fractures in the extra life years, while these costs were not included in the model of Cobiac et al [2].

1.7 Uncertainty analysis

Table 1: Parameters that were varied in the uncertainty analysis.

Parameter	***Values***	***Uncertainty distribution***	***Source***
Fracture risk by level of BMD and age*	Variable; see appendix 2.	Normal	Data [8] provided by prof. Kanis (appendix 2).
RR falls**	0.89 (0.78-1.01)	Normal around log RR	Cochrane review [14]
Proportion hospitalised	Varies by fracture site	Beta	Dubbo study [13]
Cost-offsets	Varies by fracture site.	Gamma	Dubbo study [13]

* Only relevant for scenario via BMD

** Only relevant for scenario that links physical activity directly to fracture incidence

1.8 Results and Sensitivity Analysis

The intervention was already cost-saving without the added fracture prevention-effect, which adds a modest 134 DALYs (+0.6%) and cost-savings of $4.5 million (7%) (Table 2).

Table 2: Results of the base case scenario.

Base case scenario (25-60 year olds)	*Without fractures*		*With fractures*		*Additional effect fractures*	
	Median	95% UI	Median	95% UI	Median	95% UI
years of life saved	14,000	10,000 – 19,000	14,000	10,000 – 19,000	51	-1 – 105
DALYs averted	22,000	16,000 – 29,000	22,000	16,000 – 29,000	134	-5 – 277
cost intervention (million $)	13	11 – 16	13	11 – 16	0	-
net costs (million $)	-68	-18 – -137	-68	-18 – -137	-4.5	-13.2 – +1.1
ICER with cost-offsets (million $)	Dominant (cost saving)		Dominant		N/A	
ICER without cost-offsets (million $)	Dominant		Dominant		N/A	

Note: UI = uncertainty interval

Table 3: Sensitivity analysis (median values)

	Additional DALYs	*Additional Cost-offsets (million $)*
Base case	134	4.5
25% decay BMD (no change fall propensity)	177	5.1
10% decay BMD (no change fall propensity)	639	13.0
50% decay BMD but 10% permanent	2,210	50.7
25% decay fall propensity	311	9.4
10% decay fall propensity	1375	33.3
25% decay in physical activity (and BMD and fall prop.)	392	10.6
Age 60-90, effect half that in those aged 25-60	893	17.3
No discounting	26	0.3

* 50% decay in PA, BMD and fall propensity

Three factors have a major influence on the results: the rate at which the extra bone added by PA is lost, the rate at which the falls-reducing effect of PA wanes, and the age of the target group (Table 3).

The base case scenario supposes that gain in BMD is only present for as long as the extra physical activity is maintained, and this in turn is assumed to decline by 50% per year. If this assumption is relaxed and replaced with a BMD-decay of only 10% per year, the health benefit increases almost 5-fold and cost-offsets almost 3-fold. If the falls-reducing effect of the intervention were to last longer, this would have about twice as much effect. Targeting people at older ages significantly increases the benefits both in terms of health and reduced health care spending, even if lower effects on physical activity are assumed.

That the age of the target group also matters is not surprising as most fractures occur in old age. Table 4 shows the effects of adding the separate pathways (bone mineral density and falls) for different age cohorts if the intervention-effect were to affect all ages equally in terms of time spent physically active. Relative to the effects via CVD, diabetes and cancer, the fracture prevention adds a modest 5% of the health effects. But this differs widely by

age: under the assumptions in the model, the extra benefit rises with age to 50% over the effect of the other diseases at that age cohort. It also shows extra health care savings. This stresses the importance of physical activity for the health of the elderly (though it does not show how to activate this group).

Table 4: Effects of adding fracture-reducing effect of physical activity to interventions (base case scenario among 25-60 year olds extended to all ages) in DALYs by age group (men and women aggregated) and lifetime health care costs, compared to a model that contains ischemic heart disease, stroke, diabetes, breast cancer and colon cancer (of which the results are not shown in this table).

Age	*DALYs (BMD)*	*DALYs (falls)*	*DALYs (total)*	*DALYs (%)*	*HC costs over the rest of life (average p.p.)*
25-29	0	6	6	1.3%	$0
30-34	0	6	6	0.6%	$0
35-39	0	9	9	0.5%	$0
40-44	0	13	13	0.4%	$0
45-49	0	20	20	0.4%	$0
50-54	0	31	31	0.5%	-$1
55-59	5	46	50	0.8%	-$1
60-64	38	62	99	1.9%	-$2
65-69	55	97	150	3.3%	-$3
70-74	86	168	251	6.3%	-$7
75-79	136	293	424	12.9%	-$14
80-84	130	348	473	22.0%	-$26
85-89	90	280	367	30.7%	-$44
90-94	34	127	159	37.6%	-$54
95+	9	40	49	50.9%	-$51
Sum/average	582	1,544	2,127	5%	-$3

Note: DALYs (BMD) and DALYS (falls) do not add up exactly to DALYs (total) because of interaction.

1.9 Discussion

If they are intended to prevent fractures, mass media campaigns that aim to increase physical activity are most likely to be effective if they target older people. However, the evaluation of a similar campaign targeting 55+ year olds did not observe a significant change in physical activity behaviour [12]. The question how to activate older people therefore remains. At ages below 60 years, a mass media campaign that achieves an average of 38 minutes extra PA per week is estimated to result in a small reduction in fracture-associated health loss. The results of this study add little extra to the results via cardiovascular disease, diabetes and cancer presented in an earlier ACE Prevention paper [2, 11], which showed the intervention to be cost-saving even at younger ages.

Even if the intervention were to result in 38 minutes/week of extra physical activity up to age 100, the fracture-related health gain is only a quarter of the effect of screening one third of all Australian women aged 70 to 90 for low BMD and treating osteoporosis with alendronate [15], or about as much as the effect of screening plus treatment with raloxifene [16]. However the effects via fractures are only about 5% of the total health benefit of physical activity (Table 4) and the costs of screening + treatment are much higher than those of the media campaign.

Strong points of this study are that, to our knowledge, this is the first time that fractures have been included in the cost-effectiveness analysis of an intervention that promotes physical activity. Furthermore, the estimates of the effect of physical activity on BMD and fracture risks are based on meta-analyses of randomised controlled trial evidence.

This study has several limitations. The first is that the change in physical activity due to the media campaign is based on a single study that showed a marginally significant effect based on a small increase in PA in the intervention population and a larger decrease in the control population. A second limitation is that the durability of the effects on both the falls reduction and the BMD increase is very uncertain. The evidence on how long the PA-induced BMD lasts is inconclusive (Appendix 3), but does suggest that physical activity in adolescents and young adults may result in permanent BMD gains. It remains possible that interventions that increase the PA-levels of adolescents are also cost-effective. However, the time between intervention and health effect would be long so the discount rate used would significantly influence cost-effectiveness ratios. In the absence of good evidence of lasting bone-strengthening effects of physical activity among middle-aged and older adults, we conservatively assumed that physical activity reduces fracture risk only in the short term. This means that effectiveness rises rapidly with age as fracture risk is strongly age-dependent.

In conclusion, the earlier paper [11] showed a mass media campaign designed to increase physical activity to be highly cost-effective. Adding the fracture-preventing effects of physical activity improves cost-effectiveness only minimally. It also suggests that to prevent fractures, targeting older people is most (cost-)effective.

1.10 References

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1.11 Second stage filter analysis summary for media campaign to improve bone health via physical activity (appendix 1)

Cost per DALY	Strength of evidence	Equity	Acceptability	Feasibility	Sustainability	Relevance to indigenous population	'other effects' (not captured in modelling)
- cost offsets: Depends on age **+ cost offsets:** Depends on age	Weak. Large uncertainties in duration of both effects of PA on bone strength and risk of falls	Potential to increase inequities due to differential uptake by socio-economic position.	Good.	Highly feasible.	Implementation flexible; can be a one-off campaign. Long-term effects on fracture incidence uncertain.	Uncertain; fracture prevention is not a priority area for Indigenous health.	Positive: CVD, diabetes & cancer [11]. Negative:
Decision point:	Better evidence for effects on CVD and cancer.						

Policy Considerations: Mass media campaigns are considered most effective when part of a broader campaign to improve physical activity levels.

1.12 Relative risks by BMD level (appendix 2)

z-score of femoral neck

Same data as used in Johnell, O., J. A. Kanis, et al. (2005). "Predictive value of BMD for hip and other fractures." J Bone Miner Res 20(7): 1185-94.

Poisson model: 1. constant, 2. current time, 3. current age, 4. BMD z-score, 5. age x BMD z-score, 6. BMD z-score x BMD z-score
RR (95% confidence interval)

Men+Women, Outcome: hip fracture

Age	-3	-2	-1	0	1	2	3
50	3.35(2.24, 5.00)	3.25(2.24, 4.73)	3.17(2.13, 4.70)	3.08(1.95, 4.88)	3.00(1.73, 5.20)	2.92(1.51, 5.65)	2.84(1.30, 6.18)
55	3.11(2.21, 4.38)	3.03(2.23, 4.12)	2.95(2.11, 4.11)	2.87(1.91, 4.31)	2.79(1.68, 4.64)	2.72(1.45, 5.07)	2.64(1.25, 5.59)
60	2.90(2.19, 3.84)	2.82(2.23, 3.58)	2.75(2.10, 3.59)	2.67(1.87, 3.82)	2.60(1.62, 4.16)	2.53(1.40, 4.58)	2.46(1.19, 5.07)
65	2.70(2.16, 3.37)	2.63(2.23, 3.09)	2.56(2.08, 3.15)	2.49(1.82, 3.40)	2.42(1.56, 3.75)	2.35(1.33, 4.16)	2.29(1.13, 4.62)
70	2.51(2.13, 2.97)	2.45(2.27, 2.64)	2.38(2.05, 2.76)	2.32(1.76, 3.05)	2.25(1.49, 3.40)	2.19(1.27, 3.80)	2.13(1.07, 4.24)
75	2.34(2.07, 2.64)	2.28(2.14, 2.34)	2.22(2.02, 2.43)	2.16(1.68, 2.77)	2.10(1.41, 3.12)	2.04(1.19, 3.49)	1.99(1.01, 3.91)
80	2.18(1.97, 2.41)	2.12(1.99, 2.26)	2.06(1.94, 2.20)	2.01(1.57, 2.56)	1.95(1.32, 2.89)	1.90(1.12, 3.24)	1.85(0.94, 3.63)
85	2.03(1.81, 2.28)	1.97(1.85, 2.10)	1.92(1.76, 2.10)	1.87(1.46, 2.40)	1.82(1.23, 2.70)	1.77(1.04, 3.02)	1.72(0.88, 3.39)

Men+Women, Outcome: osteoporotic fracture without hip fracture

Age	-3	-2	-1	0	1	2	3
50	1.37(1.09, 1.73)	1.30(1.08, 1.55)	1.23(1.07, 1.41)	1.16(1.03, 1.31)	1.10(0.95, 1.27)	1.04(0.86, 1.25)	0.98(0.78, 1.25)
55	1.41(1.13, 1.76)	1.33(1.13, 1.58)	1.26(1.12, 1.43)	1.20(1.08, 1.33)	1.13(1.00, 1.28)	1.07(0.90, 1.27)	1.01(0.81, 1.27)
60	1.45(1.17, 1.80)	1.37(1.18, 1.61)	1.30(1.17, 1.45)	1.23(1.13, 1.34)	1.16(1.04, 1.30)	1.10(0.94, 1.30)	1.04(0.83, 1.30)
65	1.49(1.21, 1.84)	1.41(1.22, 1.64)	1.34(1.21, 1.47)	1.27(1.17, 1.36)	1.20(1.08, 1.33)	1.13(0.97, 1.32)	1.07(0.86, 1.33)
70	1.54(1.25, 1.89)	1.45(1.25, 1.69)	1.38(1.25, 1.51)	1.30(1.21, 1.40)	1.23(1.12, 1.36)	1.17(1.00, 1.36)	1.10(0.89, 1.37)
75	1.58(1.28, 1.95)	1.50(1.29, 1.74)	1.42(1.28, 1.56)	1.34(1.24, 1.45)	1.27(1.14, 1.41)	1.20(1.03, 1.40)	1.14(0.91, 1.41)
80	1.63(1.31, 2.02)	1.54(1.32, 1.80)	1.46(1.31, 1.63)	1.38(1.26, 1.51)	1.30(1.16, 1.46)	1.23(1.05, 1.45)	1.17(0.93, 1.46)
85	1.67(1.34, 2.10)	1.58(1.34, 1.88)	1.50(1.32, 1.70)	1.42(1.27, 1.58)	1.34(1.18, 1.53)	1.27(1.07, 1.51)	1.20(0.95, 1.51)

1.13 BMD decline after stopping (extra) physical activity (appendix 3)

Introduction

A PA promoting programme results in an amount of extra bone mineral density (BMD). However, once people stop the extra exercise and return to their usual PA patterns, how long can we expect the extra BMD to last? For some interventions, such as those that target younger people, the durability of the BMD-effect has a great influence on cost-effectiveness estimates. This appendix presents evidence from the literature and describes possibilities for modelling the long-term effects of PA on BMD. It is based on a quick review of the literature based on a search of PubMed and tracking references.

The long-term effect of extra bone mineral density

Clinicians know that when previously mobile people become bedridden they lose bone minerals at an astounding rate.

In a study based on a survey among older men (age >50 years) no relationship was found between BMD and physical activity over the lifetime when current physical activity was adjusted for [17]. However, there was an effect on other measures of bone strength (mid-femur total and cortical area, cortical BMC and polar movement inertia) of PA in adolescence, and also of continued PA thereafter.

Also, based on recall among postmenopausal women, moderate PA during adolescence was found to be positively correlated with BMD in postmenopausal women, but PA during later periods in life was not [18].

Furthermore, a Swedish study found that former male young athletes partially lost benefits in BMD (g/cm2) with cessation of exercise, but, despite this, had a higher BMD 4 years after cessation of career than a control group. And although exercise-induced BMD benefits were reduced after retirement from sports, former male older athletes had fewer fractures than matched controls. [19]. This suggests that the extra BMD from PA in adolescence is partly lost in the years after reducing PA, but that an important part may be retained. The lower fracture rate at old age may also be due to better balance, not BMD, and one may question whether the researchers have succeeded in removing all effects of recent physical activity. Former athletes may continue to have higher levels of physical activity after their formal career ends and failure to adequately correct for this results in confounding.

This is all about peak bone density, young ages and based on observational evidence. Among older people the BMD gained seems to be lost soon after quitting. Postmenopausal women lost 80% of BMD gained in an RCT in 13 months [20]. Another study found that all gains were lost within half a year [21] and a third concluded the same at 1 year post-intervention [22].

In contrast, in a RCT with high-impact exercise among fairly active, 35-45 year old women, the BMD effects were fully retained 3.5 years post-intervention while the muscle and strength effects had vanished [23]. In Figure 1, the lines of BMD for the intervention- and control groups run more or less parallel after the end of the trial at 18 months. The authors refer to the earlier studies and deem theirs stronger, but also note that menopause might make things look different.

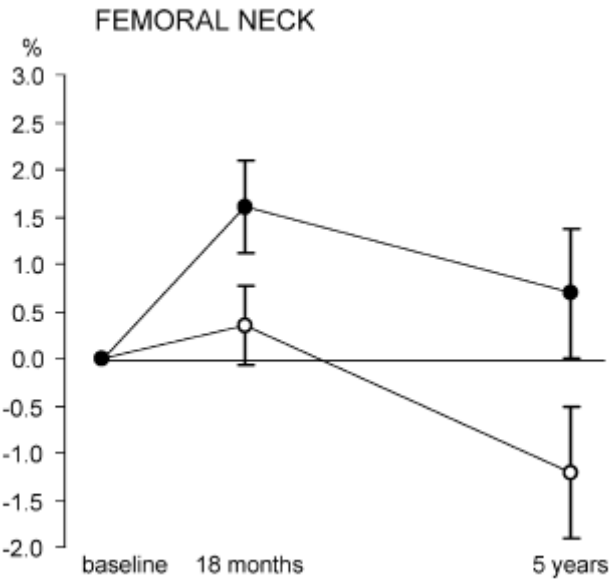

Figure: Changes in BMD of 35-45 year old women immediately after high impact exercise and 3.5 years later [23]. The closed dots are the intervention group, the open ones for the control group.

Possible modelling solutions

Our model takes into account a 'decay factor' that reduces the extra BMD at an exponential rate (i.e., a fixed percentage per year). One extreme is to link BMD directly to the level of physical activity and use the same decay factor as we use for physical activity (which we estimate at 50% per year, based on a meta-regression in the obesity field [24]). This leads to conservative estimates of the effects of interventions. We take this as the base case scenario.

But the evidence does not exclude the possibility that changes in BMD lag changes in physical activity by years, or even that some of the extra bone is added indefinitely. Such a permanent gain can be modelled. It is also possible to apply two separate decay factors; one for postmenopausal women and one for younger women and men. But in general modellers are advised to apply Occam's razor: the explanation of any phenomenon should make as few assumptions as possible. In the sensitivity analysis we relaxed the assumption that BMD is directly linked to current levels of physical activity.

Tentative conclusion

The evidence is limited and seems inconsistent unless we accept that the effects are different pre-menopausally versus post-menopausally. Physical activity in adolescents and young adults seems to result in (some) permanent BMD gains, while exercise among post-menopausal women might only provide temporary BMD increases which are quickly lost upon detraining.

For our model, BMD loss after physical activity might be anything between 'all lost within a year' to 'all gains retained until the end of life'. A more formal review and meta-analysis can be done, but may run into difficulties regarding the format of reported outcomes and is unlikely solve the whole problem as the uncertainty is mostly around the effect of menopause. There seems to be insufficient evidence yet to perform a meta-regression.

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