Parkinsonisme onder wit sendingpersoneel in Malawi: 1889-1989

Parkinsonism amongst white missionary workers in Malawi: 1889-1989

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ABSTRACT

The study was launched in order to investigate a suspected increase of Parkinsonism among white Dutch Reformed Church (DRC) missionary workers in Malawi. Since the founding of a DRC mission in 1889 and up to 1989, 562 adults joined the Mission in Central Malawi. Eleven cases of Parkinsonism occurred in this population (incidence of 1,96%): 3 women and 8 men. Only 2 were diagnosed while in Malawi, aged 55 and 59 years. In the rest, the diagnosis was made 8-50 (mean 21,4) years after departure from the country, at the mean age of 63,7 (43-80) years. The mean length of illness was 9 (4-18) years. Four patients are still alive today, with a mean survival time of 15,2 (7-19) years. The mean length of stay in Malawi, for all patients, was 20,8 (4-45) years. The vast majority of international Parkinsonism surveys record prevalence, and could thus not be compared with our incidence figures. In view of this, and our relatively small sample, it was not possible to show an overall increase of Parkinsonism among DRC missionaries. However, when individual stations were compared, a statistically significant increase of cases were found on 3, situated within a common radius of 50 km. No reason for this phenomenon is evident, and we suggest that an epidemiological study of Parkinsonism among indigenous Malawians in the Central District may reveal interesting collaborative information.

INLEIDING

Die vermoede dat daar onder wit sendingwerkers van die Nederduits Gereformeerde Kerk (NGK) in Malawi, en op sekere sendingstasies in die besonder, 'n verhoogde voorkoms van Parkinsonisme is, het gelei tot hierdie navorsing, waartydens 'n ondersoek geloods is na die voorkoms van Parkinsonisme in hierdie groep, oor die tydperk 1889-1989. Die navorsing is gebaseer op rekords van die NGK, veral dié van die Kaapse Sendingkantoor, notules van die Malawi Sendingraad, sowel as uitgebreide persoonlike navraag by familie van sendingwerkers en betrokke geneeshere. Een van die outeurs (C.J. Blignaut) was van 1956-1997 geneesheer op Nkhoma-sendingstasie, G.M. Retief was skoolhoof (1958-1998) in Malawi, terwyl F.P. Retief en G.M. Retief daar gebore is.

NGK-SENDINGAKSIE IN MALAWI[1,2,3,]

Die Kerk van Skotland het in 1875, onder leiding van dr. R. Laws, die eerste Presbiteriaanse sendingaksie in die destydse Nyassaland geloods. Dit is interessant om daarop te let dat die eerste Suid-Afrikaanse sendelinge na Malawi vier Xhosas was wat Laws as tolke vergesel het. In samewerking met die Skotte het di. A.C. Murray en T.C.B. Vlok die NGK-sendingaksie in 1889 te Mvera geloods. Die sending het flink gegroei totdat daar in 1960 14 stasies met 140 werkers was wat 'n wye spektrum aktiwiteite (o.a. onderwys, geneeskunde, landbou, tuisnywerhede, volwassene-onderrig, ens.) bo en behalwe die evangelisasie, gedek het. Mediese werk is van die vroegste tye af deur sendingwerkers met beperkte kennis gedoen, maar het formeel in 1900 begin, toe die eerste dokter (W.A. Murray) te Mvera gestasioneer is. Mettertyd het die hospitaal op Nkhoma (hoofstasie) as mediese sentrum oorgeneem.

Ná onafhanklikwording van Malawi (1962) is die aktiwiteite van die NGK volledig oorgeplaas na inheemse bestuur – en het die NGK sendingaksie amptelik tot 'n einde gekom. Voortgesette steun uit Suid-Afrika het wel voortgeduur, maar verskraal. Met die Eeufeesviering in 1989 was daar nog 31 NGK-

gesteunde personeel werksaam op vier oorblywende "sendingstasies".

VOORKOMS VAN PARKINSONISME

Oor die 100 jaar – 1889-1989 – het 562 volwasse wit sendingwerkers (361 vroue, 201 mans) uit Suid-Afrika na Malawi gegaan, en is 274 kinders (132 dogters, 142 seuns) daar gebore. Elf persone het Parkinsonisme ontwikkel (insidensie van 1,96%), van wie vier nog leef (**Tabel 1**). Hierdie statistiek word verder ontleed.

Die diagnoses deur bekwame geneeshere gemaak op die basis van algemeen aanvaarde kliniese riglyne, is sover moontlik deur die outeurs retrospektief bevestig. Daar was agt mans (72,7 %) en drie vroue (27,3 %) wat die siekte ontwikkel het, en die ouderdomme (by diagnose) het gewissel tussen 43-80 jaar (gemiddeld 62,8 jaar). By slegs twee persone (JHR, DdT) is die diagnose in Malawi gemaak. By die res is die diagnose tussen 8 en 50 jaar (gemiddeld 21,4 jaar) ná hulle vertrek uit Malawi gemaak. Vier persone lewe nog, 7-19 jaar ná diagnose (gemiddeld 14,2 jaar), terwyl sewe 4-18 jaar ná diagnose (gemiddeld 9,0 jaar), op 'n gemiddelde ouderdom van 75,1 jaar (63-88 jaar), gesterf het. Die oorlewendes se ouderdomme wissel tussen 61-80 jaar (gem. 70,3 jaar). Hulle totale verblyf in Malawi wissel tussen 4 en 45 jaar (gemiddeld 20,8 jaar). Vier was predikante, twee was huisvroue, twee boere, twee onderwysers en een 'n boekhouer.

Bereken volgens die jaar van aankoms, was die voorkomssyfer van Parkinsonisme 0 % voor 1900, 3,9 % in die tydvak 1901-1920, 2,9 % vir 1921-1940, 1,5 % vir 1941-1960 en 1 % na 1960. Bereken as 'n jaarlikse voorkoms per 100 000 van die bevolking, is die geheelgemiddeld 19,6 (194,2 vir 1901-1920, 147,1 vir 1921-1940, 73,1 vir 1941-1960 en 35,7 ná 1960).

In **Tabel 2** word die aantal Parkinsonisme-gevalle per stasie/dienspunt uitgedruk as 'n persentasie van die totale personeellading oor 100 jaar. Drie stasies trek die aandag met voorkomssyfers van meer as 10 %, nl. Mphunzi (12 %), Chongoni (27,3 %) en Chitundu (40 %). Mchinji se voorkoms was 6,3 % en Mvera 4 %; die res was laer as 4 %.

In **Tabel 2** word die voorkoms ook uitgedruk as die aantal "Parkinsonisme-geassosieerde" personeeljare per stasie, in verhouding tot die totale aantal personeeljare. Weereens toon Mphunzi (19,4 %), Chongoni (49,5 %) en Chitundu (8 %) die hoogste voorkoms. Mchinji is tans 1,0 %, en Mvera 3,6 %; Kasungu is redelik hoog met 7,0 %.

Hierdie interstasie-voorkomsvergelyking word egter vertroebel deur die relatiewe klein personeelgetalle by sommige stasies, en, baie belangrik, deur die feit dat dieselfde personeel op verskeie stasies werksaam was. Om vergelykbare statistiek te verkry, sou dit meer korrek wees om die aantal Parkinsonisme-pasiënte wat by 'n betrokke stasie werksaam was, telkens te vergelyk met die voorkoms van Parkinsonisme by daardie personeelgroep wat nooit by die betrokke stasie gewerk het nie. So 'n vergelyking word in **Tabel 3** gemaak, waar die beduidendheid van elke stel vergelykings ook statisties bereken is (statisties beduidendheid word aangedui deur 'n p-waarde kleiner as 0,05 %):** By statistiese ontleding is, soos gepas, telkens van die X^2-analise of Fisher Eksakte-analise gebruik gemaak. Die samewerking van me. G. Joubert, Dept. Biostatistiek, Fakulteit Gesondheidswetenskappe, Universiteit van die Vrystaat, word met waardering erken. slegs die stasies Mphunzi ($p = 0,010$), Chongoni ($p < 0,001$) en Chitundu ($p = 0,003$) is beduidend.

Wanneer kombinasies van stasies getoets word, toon Mphunzi, Chongoni en Chitundu ($p = 0,001$) gesamentlik 'n statisties beduidende verhoogde voorkoms van Parkinsonisme.

TABEL 1: PARKINSONISME – GEVALLE

Persoon	M/V	Geb.	Beroep	Stasies	Jare	Diagn.	Oud	Verloop	Sterf	Oud	Wanneer diagnose gemaak is
1. A.G.M.	M	1872	Predikant	Mlanda	14	1930	58	Dood ná 5 jaar	1935	63	Diagn. 15 jaar na vertrek
2. J.H.R.	M	1886	Predikant	Mlanda Malingunde Mchinji Mvera Nkhoma Mphunzi	2 3 1 35 6 4 19	1945	59	Dood ná 12 jaar	1959	71	Diagn. in Malawi
3. D.d.T.	M	1895	Boer	Nkhoma Chongoni	2 43 45	1950	55	Dood ná 16 jaar	1966	71	Diagn. in Malawi
4. W.U.	V	1890	Huisvrou	Mphunzi Mvera Chitundu Malingunde	5 9 18 2 2	1958	68	Dood ná 10 jaar	1968	78	Diagn. 29 jaar ná vertrek
5. A.D.	M	1902	Boekhouer	Mvera	4	1980	78	Dood ná 10 jaar	1970	88	Diagn. 50 jaar ná vertrek
6. A.H.T.	M	1907	Boer	Nkhoma	34	1972	65	Dood ná 4 jaar	1976	69	Diagn. 8 jaar ná vertrek
7. P.J.B.	M	1906	Onderwyser	Chintembwe Chitundu Nkhoma	2 2 24 20	1986	80	Dood ná 6 jaar	1992	86	Diagn. 22 jaar ná vertrek
8. J.D.H.S.	M	1921	Predikant	Dzenza Mvera Mlanda Mphunzi Mlanda Nkhoma	2 1 4 29 2 2 8	1994	73	Lewe nog, 7 jaar	-	80	Diagn. 20 jaar ná vertrek
9. D.I.R.	V	1931	Huisvrou	Chongoni Kasungu	3 10 13	1982	51	Lewe nog, 19 jaar	-	70	Diagn. 21 jaar ná vertrek
10. H.P.	M	1931	Predikant	Kongwe Chongoni	1 5 6	1988	57	Lewe nog, 13 jaar	-	70	Diagn. 18 jaar ná vertrek
11. A.J.	V	1940	Onderwyser	Nkhoma	7	1983	43	Lewe nog, 18 jaar	-	61	Diagn. 10 jaar ná vertrek

Fig. 1

TABEL 2: PARKINSONISME –VOORKOMS PER STASIE

Stasie	Tot. Personeel	Parkins.-gevalle	%	Tot. perso-neeljare	Parkins. personeeljare	%
Nkhoma	352	6	1,7	2 485	75	3,0
Kongwe	96	1	1,0	559	1	0,2
Mvera	101	4	1,0	584	20	3,6
Mlanda	105	3	2,9	499	22	4,4
Malingunde	82	2	2,4	292	5	1,7
Chintembwe	50	1	2,0	219	2	0,9
Dzenza	42	1	2,4	181	2	1,1
Kasungu	37	1	2,7	142	10	7,0
Mphunzi	25	3	12,0	134	26	19,4
Lilongwe	28	0	0	110	0	0
Chongoni	11	3	27,3	103	51	49,5
Mchinji	16	1	6,3	95	1	1,0
Malembo	22	0	0	81	0	0
Chitundu	5	2	40,0	50	4	8,0
Blantyre	9	0	0	39	0	0
Livlezi	6	0	0	19	0	0
Zomba	7	0	0	14	0	0
Totaal	**976**	**28**	**2,9**	**5 557**	**219**	**3,9**

Volgens **Fig. 1** is dit opvallend dat hierdie drie stasies geografies na aan mekaar geleë is, binne 'n radius van ongeveer 50 km.

BESPREKING

Klinies-epidemiologiese aspekte

In die literatuur word die voorkoms van Parkinsonisme by mans en vroue as ongeveer gelyk[4,5] aangedui, alhoewel Harada *et al.*[6] 'n verhoogde vroulike voorkoms, en Onuaguluchi[7] 'n verhoogde manlike voorkoms aantref. In ons klein reeks het mans ook oorheers. Parkinsonisme kom kenmerkend by laat middeljariges en bejaardes voor en het 'n variërende maar tipies verlengde oorlewingstyd van sewe jaar of langer. Parkinsonisme verkort wel lewensverwagting: Nobrega *et al.*[8] rapporteer 'n normale oorlewingsvoorkoms in die eerste vyf jaar, maar 25 % verhoogde mortaliteit ná 10 jaar, en 33 % ná 15 jaar ná diagnose. Die pasiëntedata in hierdie navorsing val dus binne hierdie breë parameters.

Ten einde te bepaal of die voorkoms van Parkinsonisme onder die navorsingsgroep in Malawi verhoog het, is dit vergelyk met bevindings van outeurs wat uitvoerig hieroor gepubliseer het. Gegewens uit Afrika is uiters beperk. Ongelukkig berus feitlik alle beskikbare opnames verder op bepalings van *prevalensie* (voorkoms in 'n spesifieke bevolkingsgroep op 'n bepaalde tydstip) en nie op 'n langtermyn-*risiko-bepaling* soos deur ons uitgevoer nie. Omdat getalle in die Malawi-projek klein was, het prevalensiebepalings op vasgestelde tye gedurende die 100 jaar onprakties geblyk. Dit is dus nie moontlik om die aansienlike internasionale statistiek, uitvoerig saamgevat deur o.a. McKeigue en Marmot,[9] met ons resultate te vergelyk nie. Een vergelykbare projek is wel deur Rajput *et al.* oor 'n tydperk van 13[10] jaar uitgevoer, wat 'n jaarlikse insidensie van 20,5 per 100 000 van die bevolking toon. Wanneer 'n dergelike analise by ons projek gedoen word, wissel dit aansienlik oor die 100-jaartydperk met 'n progressief dalende tendens (per tydvakke van 12-28 jaar bereken), maar die totale syfer van 19,6 per 100 000 van die bevolking kom nou ooreen met dié van Rajput *et al*.

'n Verhoging van die totale aantal Parkinsonisme-gevalle in die Malawi-sending kan dus nie bevestig word nie, maar daar was 'n statisties beduidende vermeerdering van Parkinsonisme op drie stasies, nl. Mphunzi, Chongoni en Chitundu. Terselfdertyd is die outeurs egter daarvan bewus dat konvensionele statistiek soms onverwagte probleme (en dus twyfelagtige resultate) kan oplewer wanneer getalle relatief klein is, soos in ons geval, en veral by analises gegenereer deur *post hoc*-kommer oor spesifieke probleemsituasies.[11] Huidige gevolgtrekkings word dan ook met omsigtigheid teen hierdie agtergrond aangebied.

Etiologie

Die patogenese van Parkinsonisme word nie volledig verstaan nie. Die siekte word histologies gekenmerk deur sellulêre degenerasie in die brein se substantia nigra, afname in neuromelanien en teenwoordigheid van Lewy-liggame – afwykings wat in ligter graad ook by normale bejaardes aangetref kan word. Die siekte het waarskynlik 'n multifaktoriese patologie met die normale verouderingsproses as basiskomponent. Daar is geruime tyd vermoed dat oorerflikheid 'n rol speel, maar onlangse navorsing (veral by identiese tweelinge) skyn genetiese invloede grotendeels uit te skakel.[12,13] Daar is wel bewys dat Parkinsonisme by swart volke en Asiërs 'n skaarser siekte is as by persone van Europese afkoms – ook in die VSA.[4,6,14,15] Presipiterende omgewingsfaktore soos infektiewe agense, toksiese stowwe, medikamente en trauma speel waarskynlik 'n etiologiese rol, en veel is reeds hieroor gepubliseer.[10,12]

Die konsep *arteriosklerotiese Parkinsonisme* as entiteit word hedendaags betwyfel. Sogenaamde *idiopatiese Parkinsonisme*, waar geen van bovermelde veroorsakende faktore aantoonbaar is nie, bly steeds die volopste variëteit van die siekte.[12]

Verskeie outeurs meen dat epidemiologiese studies op 'n kohort-effek by die ontwikkeling van Parkinsonisme dui, met variërende intervalle tussen impak van die veroorsakende stimulus, en die ontstaan van die siekte.[9] So 'n tydsverloop, van onbekende omvang, sou die dalende voorkomssyfer by ons pasiënte kon verklaar: 3,9 % en 2,9 % vir 1901-1920 en 1921-1940 onderskeidelik, vergeleke met 1,0 % ná 1960. Dit impliseer verder dat nog meer Parkinsonisme-gevalle in die toekoms uit die groep verwag kan word.

TABEL 3: PARKINSONISME – VOORKOMS

Stasies	Personeel ooit daar	Gevalle	Personeel nooit daar nie	Gevalle	*P-waarde
Nkhoma	352	6 (1,7%)	210	5 (2,4%)	0,345
Mvera	101	4 (4,0%)	461	7 (1,5%)	0,117
Mphunzi	25	3 (12,0%)	537	8 (1,5%)	0,010
Chongoni	11	3 (27,3%	551	8 (1,5%)	< 0,001
Mlanda	104	3 (2,9%)	457	8 (1,8%)	0,439
Chitundu	5	2 (40,0%)	557	9 (1,6%)	0,003
Mphunzi, Chongoni, Chitundu	38	7 (18,4%)	524	4 (0,8%)	0,001

GEVOLGTREKKINGS

Die 11 Parkinsonisme-gevalle wat in ons navorsing geïdentifiseer is, was oënskynlik almal idiopaties, in soverre dat geen primêre oorsaak vasgestel is nie. Daar was 'n hoër voorkoms by drie stasies, geografies na aan mekaar geleë, binne 'n radius van ongeveer 50 km. Die betrokke persone was nie aan bekende veroorsakende geneesmiddels blootgestel nie.

Geografiese lokalisering, ontkoppel van ras, is in die VSA beskryf waar die noordelike gebiede 'n hoër voorkoms van Parkinsonisme as die suide toon, maar verskille (steeds onverklaard) was gering, vergeleke met die Malawi-ondervinding.[4,15] Rajput het in Saskatchewan, VSA, gelokaliseerde vroeë-aankoms-Parkinsonisme beskryf wat oënskynlik aan die drink van fonteinwater gekoppel kon word.[16] Indien die heersende siening korrek is, nl. dat die patogenese van die siekte berus op presipiterende omgewingsfaktore wat, in kombinasie met die normale verouderingsproses, Parkinsonisme aanbring, sou 'n mens kon postuleer dat so 'n omgewingsfaktor in die Mphunzi-/Chongoni-/Chitundu-area aanwesig kan wees. 'n Gerigte epidemiologiese ondersoek in dié gebied is na ons mening aangewese. Reef[17] het in 'n Suid-Afrikaanse prevalensie-studie getoon dat swartes 'n laer voorkoms van Parkinsonisme as wittes toon, maar ons is nie van enige dergelike ondersoeke in Malawi bewus nie.

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Bone mineral density and menstrual function in adolescent female long-distance runners –

A prospective comparative study of bone structure and menstrual function in adolescent female endurance athletes from five secondary schools in Pretoria.

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Key words: Female adolescent endurance runner; premature osteoporosis; bone mineral density.

ABSTRACT

Background. In recent years, endurance running as a sport has become very popular. This trend has led to the identification of specific problems during the female athlete's life, especially with regard to reproduction, delayed sexual maturation, menstrual abnormalities and early osteoporosis.
Methods. Bone mineral density (BMD) and menstrual function were compared between a group of long-distance female adolescent runners (N=17) from five schools in Pretoria and an age-matched inactive control group of adolescents (N=18). Groups were matched for body height, mass index (BMI 18 to 25) and eating habits. The SAHARA Clinical Sonometer was used to measure BMD on the calcaneus. Menstrual function was denoted by onset of menarche, duration of menstrual periods (days) and number of menstrual periods per year.
Results. Baseline BMD was significantly higher in the athletic group: mean = 0,6126 g/cm^3 and SD = 0,1217, versus non-athletes: mean = 0,5329 g/cm^3 and SD=0,0733 (p = 0,0228). There was a significant delay in the onset of menarche in the athletes: mean = 14,873 and SD = 1,37798, in comparison to the non-athletes: mean = 13,468 and SD = 1,2194 (p = 0,0030). The athletes had a significantly higher incidence of menstrual abnormalities (p = 0,005).
Conclusions. BMD at the focus of strain for running (the legs) is higher in endurance adolescent female runners. Endurance runners have a significantly higher incidence of menstrual abnormalities.

Background

Women of all ages are becoming increasingly involved in strenuous athletic activity for fitness and/or competitive reasons. The growth in the participation of women in sport occurred as a result of legislation passed in the USA in 1972 called "Title IX". This states that any school receiving federal assistance must offer equal athletic opportunities to men and women (participation, scholarship, money and athletic benefits).

Despite the numerous benefits of physical activity, specific problems might occur in the female athlete's life, especially with regard to reproduction. These include delayed sexual maturation in the growing athlete and abnormal or absent menstrual cycles in the mature woman, with potential adverse effects on the foetus of the pregnant woman. Other problems are eating disorders and skeletal abnormalities, including reduced bone density (BMD), scoliosis, stress fractures and failure to reach peak bone mass.[1]

The BMD might be influenced by several variables, such as current menstrual status, menstrual history, body mass, functional loading, family history of osteoporosis, nutritional status, training intensity and frequency, and calcium balance.[2,3] These problems are encountered by female athletes who take part

in endurance running, rowing, gymnastics, ballet, swimming, basketball and cycling.

The decrease in BMD can predispose the female athlete to an increased risk of stress fractures. BMD loss is a silent process and the athlete is usually unaware that a problem exists until a related injury (such as a stress fracture) occurs.

In 1992, the American College of Sports Medicine published an article about the so-called "Female Athletic Triad", describing the large increase in the prevalence of three disorders presenting together in the athletic female. This triad consists of disordered eating, delayed menarche/menstrual abnormalities and osteoporosis. It is the major health risk factor facing women athletes today. The aetiology of this triad is multifactorial, with risk factors including nutrition, menstrual status, training intensity and frequency, body mass, family history of osteoporosis, calcium balance and psychological/ physical stress. Medical management of this triad requires a multidisciplinary approach, with the key being early diagnosis and therapy.[4]

The consequences and long-term effects of delayed puberty on young athletes are not completely clear. Bone mass accretion, which normally occurs during adolescence (18 to 25 years,[4] while other authorities claim 16 to 18 years[5]), is compromised in girls maturing late due to the negative impact on gonadal function. Maximising peak bone mass in the adolescent woman is of the utmost importance, as it is the time when athletes should be storing bone for the inevitable loss in later years. The two to three years of the pubertal growth spurt are accompanied by deposition of 60% of the final bone mass, and any dietary inadequacy and high exercise intensities at this time could severely alter bone formation.[3]

There have been conflicting results concerning bone mass accretion (measured by the BMD) and menstrual abnormalities in different studies over the past few years. There are no long-term follow-up studies of former amenorrhoeic athletes that enable the determination of whether normal BMD can be attained following several years of abnormal menses or use of oral contraceptives. Martin et al. state that the bone mass of the lumbar spine of women with a history of oligomenorrhoea/amenorrhoea might never reach that of women who have had regular menstrual cycles.[6] A recent study by Drinkwater et al. reports that, after six to 10 years of amenorrhoea, oligomenorrhoea or oral contraceptive use, even previously amenorrhoeic athletes did not show significant improvements in vertebral BMD values. Thus, these findings suggest that oligomenorrhoea is as detrimental to the lumbar spine as amenorrhoea.[7] Moreover, the long-term effects of amenorrhoea on fertility are still unclear, although there are data suggesting that the reproductive deficiencies associated with amenorrhoea are reversible when the problem is treated.[8]

Most of the data come from the USA and it was interesting to see whether these findings would apply to South Africa. In recent years, long-distance running as a sport (primary and secondary school level) has become more popular, and children as young as eight years are practising and competing all year round.

Methods

Eighteen female adolescent endurance runners, aged between 16,083 and 18,25 years, were recruited between February and May 2001. Twenty-one female adolescents (non-athletes) aged 17,167 to 18,250 years were recruited as controls (three had to be excluded due to the current use of oestrogen for acne). Both the athletes and non-athletes were matched for weight (BMI between 18 and 25) and length and a reasonable nutritional status was maintained in both groups.

The baseline investigation included a questionnaire (including menstrual history), weight, length and ultrasonographic (SAHARA) measurement of the right calcaneus.

Definitions

Endurance (long-distance) athlete: An athlete who does endurance running for >1_ hours per day for 5 days per week for 11 months per year.
Menarche: Age at first menstrual cycle
Delayed menarche: No occurrence of menstruation before the age of 16 years.
Eumenorrhea: 10 to 13 menstrual cycles per year.
Oligomenorrhea: 4 to 9 menstrual cycles per year.
Amenorrhea: 0 to 3 menstrual cycles per year.

Study population

All white female students between 16 and 18 years attending schools in the municipality of Pretoria, Gauteng (Republic of South Africa). Black and Asian students were not excluded from the study, but none responded to the request for volunteers.

Inclusion criteria

1. Consenting adolescent female scholars between 16 and 18 years in Pretoria, Gauteng (Republic of South Africa).
2. Female scholars of all ethnic groups were included (no African or Asian athletes volunteered for testing).
3. Endurance runners who had started practising before the onset of menarche and trained for 11 months a year and for 5 days a week and >1_ hours a day.
4. Controls were recruited who did not participate in any form of organised sport.
5. Maintenance of reasonable nutritional status.
6. Body mass index of between 18 and 25 – to avoid outlier bias, the students could not differ too widely in height and weight.

Exclusion criteria

1. Use of oral contraceptives or use of hormonal treatment (i.e. for acne)

in the previous six months.
2. Drug abuse (including marijuana).
3. Smoking.
4. No parental consent.
5. Delayed menarche.

Sample size calculation

The primary efficacy variable is BMD with normal values (0,5: 0,7 g/cm^3) and hence a SD = 0,05. In order to detect a difference of 0,05 g/cm^3 in the BMD between the athlete and non-athlete groups, a sample of 17 subjects per group will have 80% power when tested two-sided at the 0,05 level of significance. The sample size calculations therefore do not depend on sampling population size.
Software used: nQuery Advisor Release 4.0, Statistical Solutions Ltd., Cork, Ireland.
Author: Janet D Elashoff, May 2000.

Written informed consent was obtained from every participant, as well as from one parent (mother or father). The Protocol was approved by the Ethical Committee of the Pretoria Academic Hospital and the Faculty of Health Sciences of the University of Pretoria.

Results

The baseline BMD was statistically significantly higher in the athletic group, with a p-value = 0,0278. This might be due to the fact that the athletes in this study had been practising for a relatively short period (mean period = 6,5490 years, SD = 2,3819).

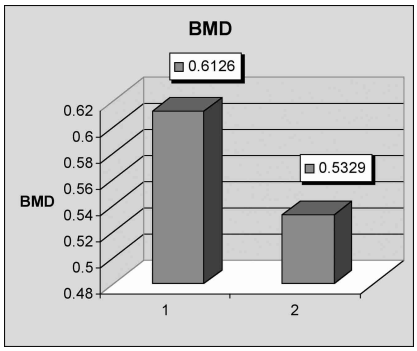

Figure I: Comparison of BMD in athletes (N=17) and controls (N=18) (g/cm2) (p = 0,0278)

There was a statistically significant difference in the onset of menstruation (menarche) between the two groups, thus suggesting a later sexual maturation in endurance athletes.

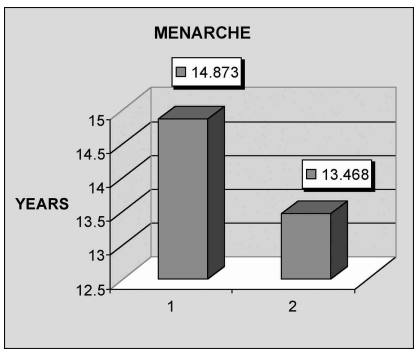

FIGURE II: Comparison of onset of menarche (years) in athletes (N=17) and controls (N=18) (p = 0,0030)

In correspondence with other studies, the expectation was to find a decrease in the menstrual flow (as measured by the menstrual period), but, surprisingly, there was no difference between the two groups. This is probably due to the fact that, even if athletes experience oligomenorrhoea or amenorrhoea (number of cycles per year), they might still have normal periods (number of days per cycle).

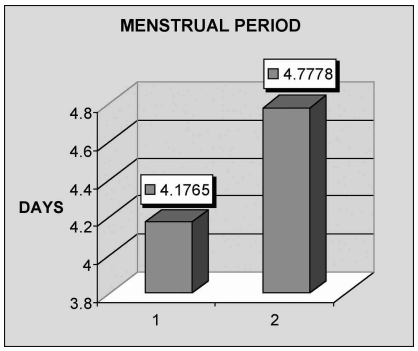

FIGURE III Comparison of menstrual period (days) in athletes (N=17) and controls (N=18) (p = 0,1875)

There was a significant difference in the menstrual function (p-value=0,005). In the athletes it was 41,2% eumenorrhoea, 35,3% oligomenorrhoea and 23,5% amenorrhoea, while in the non-athletes it was 88,9% eumenorrhoea, 11,1% oligomenorrhoea and 0% amenorrhoea. This is in accordance with other studies done elsewhere in the world.

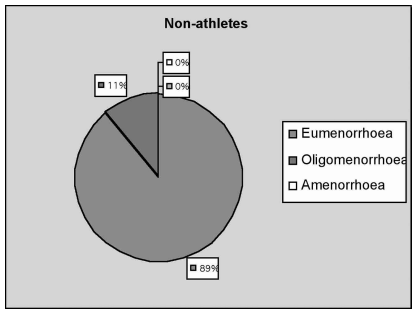

FIGURE IV: Menstrual function in athletes (N=17) and controls (N=18).

Conclusions

BMD at the focus of strain for running (the legs) is higher in adolescent female endurance runners when compared to age- and BMD-matched controls (whose training was started before the onset of menarche). When endurance runners are compared to controls in relation to their menstrual history, they display a significantly higher incidence of menstrual abnormalities, as denoted by a delay in the onset of menarche and a decreased number of menstrual cycles per year.

This study only explores exercise as the single most important factor, which is not necessarily true. Other reasons for the differences between BMD and menstrual function could be hormonal, dietary, socioeconomic, time of exercise before menarche and calcium balance. This warrants further research.

Recommendations

Identify the runners at risk for developing menstrual abnormalities. If abnormalities are present, decrease the intensity and amount of practising time, as it might have detrimental effects on their fertility and lead to early osteoporosis. These adolescent runners must be monitored carefully to help them attain peak bone mass (95% of maximum density is reached by the age of 18 years) and still perform well in their sport.

Coaching should involve a team approach, with the coach, biokinet-

icist and psychologist all available to support the long-distance trainee. Parents need to be able to identify the symptoms of injuries and menstrual abnormalities early to avoid the child losing self-confidence and performing sub-optimally.

Randomised double-blind placebo controlled trials for safe training volumes, intensities and menstrual cycle regularity, with regard to their influence on BMD in the female athlete, are needed to prevent an unnecessary decrease in performance as well as to minimise long-term health problems.

It is believed that further studies assessing the long-term health consequences of athletic amenorrhoea are essential.

Limitations of the study

This study was not sponsored and, because of financial constraints, the SAHARA measurement (ultrasound of the heel) was used instead of DEXA. DEXA (Dual Energy X-ray Absorptiometry) is still the gold standard of BMD evaluation and might reveal the impact of oestrogen deficiency earlier and more accurately than ultrasound of the heel.

Only calcaneal BMD was done (SAHARA). It would have been better to do BMD on spinal trabecular bone (DEXA) as well. Ultrasonographic measurements (SAHARA) are good and reliable, but it is known that exercise could positively influence the results of this method. Clearly defined conclusions regarding the place of quantitative ultrasound still need to be established.

All the subjects had undergone menarche, although this was slightly delayed in the trained individuals. The duration of exposure to possible hypo-oestrogenemia is therefore limited and might explain why only positive effects of exercise are noted in the study subjects.

This study demonstrates a beneficial effect in weight-bearing exercise in relationship to the BMD in female **adolescent** runners, but does not address the long-term effect of constant training on the weight-bearing bones.

Only Caucasian girls were used and it is therefore not clear whether these findings will apply to other groups. Black and Asian students could not be recruited. This necessitates a further study to include these students. No blood tests were done to assess the hormonal status of the athletes.

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