Skin diseases among schoolchildren in Africa

Arjan Hogewoning

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Proefschrift

ter verkrijging van
de graad van Doctor aan de Universiteit Leiden,
op gezag van Rector Magnificus prof.mr. P.F. van der Heijden,
volgens besluit van het College voor Promoties
te verdedigen op donderdag 13 december 2012
klokke 15:00 uur

door

Adriaan Anne Hogewoning
geboren te Dordrecht
in 1960

The printing of this thesis was financially supported by
African Tiger Holding LTD. Accra, Ghana
The Koornzaayer Foundation, The Netherlands

ISBN: 978-94-6191-497-2

Cover: In Zicht Grafisch Ontwerp
Layout: In Zicht Grafisch Ontwerp
Print: Ipskamp Drukkers BV, Enschede

Promotiecommissie:

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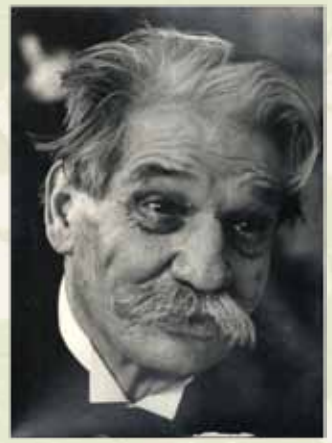

Photograph of Dr. Albert Schweitzer, by Maria Austria
and given to the author by Pieter M.Mentzel

"It was in front of three iles situated on the river Ogooué near the village of Igendja, 80 km downstream of Lambaréné, that I got the vision, one day in September 1915, that "**Reverence for life**" is the elementary principle of ethics and real humanity"
Albert Schweitzer

Aan mijn ouders

Voor Door, Pieter, Anne en Benjamin

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Chapter 1

General introduction

Children are the most vulnerable citizens in any society and the greatest of our treasures.

Nelson Mandela

Nobel Peace Prize Ceremony, Oslo, Norway, 1993

General Introduction

Skin diseases in developing countries are present in large numbers, especially among children and deserve our sincere and full attention.[1-6] They are accounting for a high percentage of visits to hospitals and primary healthcare centers and create a serious impact on health care services.[7-9] In a review of prevalence studies in children by the WHO, the prevalence of skin diseases were ranging from 21% to 87%.[10] These high figures warrant the study of morbidity, causative factors and economic costs.[1;11] Skin diseases are often considered less important in health priority programs compared with diseases that cause high mortality like tuberculosis, HIV/AIDS, meningitis or hepatitis.[2] Compared with other diseases, skin diseases have a lower mortality rate but can affect the wellbeing, quality of life and health conditions of children who already form a vulnerable group.[2]

Using a comparative assessment of disability-adjusted life years (DALY's) the World Health Organization's 2004 report on the global burden of diseases showed a total amount of 376.525.000 DALY's in Africa which was at least two times higher than in any other region in the world. For skin diseases in Africa there was a total of 902.000 DALY's (0.2% of the total burden) which was similar to that caused by several psychiatric disorders.[12]

Most of the prevalence data in Africa come from hospital or dispensary-based records and therefore are less reliable when estimating the prevalence on a national scale. Population-based prevalence figures are needed for reliable planning of national health and prevention programs. Only a few population-based studies on this subject are available.[5;9;13;14] Most of these studies have been conducted on schoolchildren. In most recent prevalence studies, conducted in sub-Sahara Africa the majority of the skin diseases found among schoolchildren are dominated by infections like tinea capitis and pyoderma.[5;10;13-20] This is a pattern found in most countries with poor socio economic circumstances. We performed several studies to gain more insight in the prevalence of tinea capitis and the causative organisms, to determine the burden of this infectious disease in communities and to identify possible strategies for prevention and treatment.

In industrialized countries the highest burden of skin diseases is formed by inflammatory diseases like acne vulgaris and eczema but recent studies from Africa reported also an increase in prevalence.[21;22] Therefore, we performed several studies focusing on "western" skin diseases and the impact of socio-economic developments on the prevalence by comparing rural versus urban schools.

The prevalence of classical tropical diseases like leprosy or filarial lymph edema is low although the socio economical impact can be enormous.[10;23-29]

This thesis focuses on three skin diseases in particular, namely, tinea capitis, eczema and acne, which diseases are discussed in some more detail below.

The most common skin diseases among children in Africa are described in **Chapter 8**. In this chapter several skin diseases among preschool children and some typical tropical diseases are presented. With this chapter (and a website to match: www.african-skindiseases.org) the author hopes to offer an easy access to basic information and pictures for healthcare workers in Africa.

The prevalence and causative organisms of tinea capitis in Africa

Tinea capitis is endemic among schoolchildren in tropical Africa.[30] Factors like over-crowding, malnutrition and climatic conditions such as heat and humidity can lead to an increase in fungal infections in tropical and semi-tropical countries.[31]

The prevalence of tinea capitis is higher among schoolchildren in rural areas due to the lack of anti-fungal treatments, poor hygienic conditions, and school and household overcrowding.[32;33]

Superficial infections of the scalp are caused by Trichophyton and Microsporum species. Those causing an endotrix infection are frequently seen in Africa. The most important causative agents are Trichophyton soudanense, Trichophyton tonsurans, Trichophyton violaceum and yaoundei. The species that cause an ectotrix infection are Microsporum audouinii, Microsporum canis and gypseum. Microsporum audouinii is frequently seen in Africa while canis is seen more often in European countries.[34;35]

Which species is causing tinea capitis is highly dependent on geography, time and social status. During the past 60 years the predominant etiologic agent of tinea capitis in the USA has changed from M. audouinii to T.tonsurans most probably due to the sensitivity of M. audouinii to griseofulvin treatment and the import of T. tonsurans by immigrants. During the late 19th and 20th centuries, M.audouini and M.canis were the most frequent etiologic agents in Western and Mediterranean Europe while Trichophyton schoenleinii was often seen in Eastern Europe.[34;36;37] In Africa the most frequently seen agents were Trichophyton soudanense, violaceum and tonsurans and Microsporum audouini. These agents are all anthropophilic and are spread rapidly in circumstances of overcrowding.[18;38-42]

The prevalence and characteristics of eczema among schoolchildren in Africa

Higher prevalences of eczema are found in developed countries like Northern Europe, North America, Japan and Australia compared with African countries. Recent studies however show a sharp increase in African countries, especially amongst infants.[5;14;21;43-49] Most of these studies are hospital based and therefore less reliable than community based studies. The questionnaire based period-prevalences are higher than the point-prevalences as measured by physical examination, which can be explained by the chronic relapsing character of eczema.[50-54]

The rising prevalence of eczema might be related to improved sanitation and reduction in childhood infections, the so called hygiene hypothesis.[53-58] Also helminthic infections have shown to induce hypo responsiveness and are negatively associated with atopy and allergy.[57;59-62] Other risk factors for the development of eczema are changes in lifestyle because of a higher socio-economic status, reduced crowding at home, changes in food consumption. Also the growing urbanization in Africa has been associated with an increased risk of eczema.[10;43;63;64]

Prevalence and risk factors of acne vulgaris in Africa

Acne vulgaris is a common skin condition in children and adolescents between the age of 10 and 18 years which is much more frequently seen in the industrialized world compared with developing countries.[21;22;65;66] Community-based studies, studying acne vulgaris in Africa are scarce. Most studies are hospital based and don't give a correct figure about the prevalence.[67] In industrialized countries this condition affects between 31% and 95% of the adolescent population while in Africa percentages of 2.8% and 8.9% are reported.[14;68;69]

With the changing socio-economic situation in developing countries, especially westernization in urban areas, it is believed that the prevalence of acne vulgaris in developing countries will increase to the level of industrialized countries.[67;70]

Aim and structure of the thesis

The aims of the thesis were:

1) *To measure the point-prevalence of different skin diseases (with special attention for childhood eczema, acne and tinea capitis) among schoolchildren in both rural and urban schools and in three different African countries (Gabon, Ghana and Rwanda).*

Between 2004 and 2007 cross-sectional studies with 4839 schoolchildren were conducted in Ghana, Gabon and Rwanda in urban and rural schools with different social economic levels (low, middle, high). All children were included in the study and were investigated by a dermatologist or a team of dermatologists.

2) *To determine causative agents for tinea capitis in Ghana and Gabon.*

In June 2004, 463 school children from 2 rural and 2 urban schools in the Greater Accra Region were fully examined by a team of dermatologists. The same happened in January 2005 in the region of Lambaréné, Gabon when 454 children in one rural and one urban school were examined. When there were clinical signs of fungal infection on

the scalp (scaling, hair loss, black dots, pustules and scars), samples were taken for analysis and transported at room temperature to the Mycology Laboratory of the Department of Dermatology of the Leiden University Medical Centre in Leiden, The Netherlands.

3) To study (socio-economic and environmental) risk factors for eczema.

A matched case-control study was performed to identify risk factors in childhood eczema. Between February and December 2005, 86 schoolchildren with moderate to severe eczema were selected at the dermatological outpatient clinics of three hospitals in Accra, Ghana by a dermatologist. For each included child with eczema, one to three controls were selected from the same school and class. All children completed a questionnaire and were skin prick tested with a panel of allergens. Blood was drawn to determine the total and allergen-specific IgE.

4) To provide information about the point and period-prevalence of eczema in West and Central Africa.

Between 2004 and 2007 cross-sectional studies with 4839 schoolchildren were conducted in Ghana, Gabon and Rwanda. To determine the point-prevalence of eczema all children in all four studies were examined by at least one dermatologist or a team of dermatologists. In Ghana the period-prevalence was measured by questionnaires adapted from the International Study of Asthma and Allergies in Childhood (ISAAC).

5) To investigate the prevalence and risk factors of inflammatory acne vulgaris in schoolchildren in Ghana.

Between between January 2006 and February 2007 a total of 1394 schoolchildren from 11 urban and rural schools in the Greater Accra Region of Ghana were screened by two dermatologists for inflammatory acne vulgaris and other skin diseases. The height and weight of the schoolchildren were measured to calculate the Body Mass Index (BMI) as a marker of nutritional status and a questionnaire was administered to each child, collecting information concerning living conditions.

Our studies were supported by the local governments and conducted in cooperation with larger studies in which atopy and parasitic infections were investigated. Our study was facilitated by the fact that the primary investigator worked as a dermatologist in these countries at the time of the investigations and had easy access to local health care facilities.

Chapter 1 provides a short introduction and defines the aims of this study.

Chapter 2 presents prevalence estimates of most skin diseases diagnosed in our studies among schoolchildren in three different countries, Gabon, Ghana and Rwanda

Chapter 3 presents the point-prevalence of tinea capitis among schoolchildren in the greater Accra region in Ghana including the most important causative fungal species.

Chapter 4 presents, like the study in Ghana, the point-prevalence of tinea capitis among schoolchildren in Gabon and the result of the determination of the fungal species and summarizes the results of the most recently published studies on tinea capitis in Africa.

Chapter 5 focuses on the point-prevalence and period-prevalence of eczema among schoolchildren in the three mentioned countries. The point-prevalence obtained by physical examination by one or more dermatologists are compared with the period-prevalence obtained by questionnaires based on ISAAC (The International Study of Asthma and Allergies in Childhood).

Chapter 6 determines allergic characteristics and identifies possible risk factors for eczema among schoolchildren in an urbanized area in Ghana.

Chapter 7 presents the prevalence of acne vulgaris among Ghanaian schoolchildren. The difference between the prevalence rates among rural and urban schoolchildren is presented as well as possible risk factors like a higher body mass index.

Chapter 8 This chapter is aimed as a practical guide for medical healthcare workers in Africa and describes the etiology, clinical signs and treatment of the most prevalent skin diseases among children in Africa and also describes some typical tropical skin diseases and some diseases among preschool children. This chapter can be accessed on internet via www.africanskindiseases.org.

Chapter 9 summarizes our results and discusses our findings in a broader perspective. The findings presented in this thesis are discussed and summarized in the Summary.

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Chapter 2

Skin diseases among schoolchildren in Ghana, Gabon and Rwanda

Accepted for publication in the International Journal of Dermatology

Arjan Hogewoning, MD [1,2,3], Abena Amoah, MSc [5],
Jan Nico Bouwes Bavinck, MD, PhD [3], Daniel Boakye, MSc, PhD [5],
Maria Yazdanbakhsh, MSc, PhD [4], Akim Adegnika, MD, PhD [4,7,8],
Stefan De Smedt, MD [6], Yannick Fonteyne, MD [6], Rein Willemze, MD, PhD [3],
Adriana Lavrijsen, MD, PhD [3]

[1] Dermatology, University of Ghana Medical School, Korle-Bu Teaching Hospital, Accra, Ghana
[2] Dermatology, King Faisal Hospital, Kigali, Rwanda
[3] Dermatology, Leiden University Medical Centre, Leiden, the Netherlands
[4] Parasitology, Leiden University Medical Centre, Leiden, the Netherlands
[5] Parasitology, Noguchi Memorial Institute for Medical research, University of Ghana, Legon, Ghana
[6] Ophthalmology, Kabgayi Hospital Rwanda
[7] Albert Schweitzer Hospital, Lambaréné, Gabon
[8] Institute of Tropical Medicine, University of Tübingen, Tübingen, Germany

Abstract

Background

Skin diseases, especially skin infections, among schoolchildren in Africa, can be a major health problem. The objective of this study was to determine the prevalence of skin diseases among children in rural and urban schools in three different African countries and to study the influence of the socioeconomic level.

M**ethods**

Cross sectional, population based studies were performed in Ghana, Gabon and Rwanda. Point-prevalences of skin diseases were estimated on the basis of physical examination by at least one dermatologist.

Results

In total, 4839 schoolchildren were seen. The overall prevalence of schoolchildren with any skin disease was high; 34.6 % and 42.0 % in two Ghanaian studies, 45.8 % in Gabon and 26.7 % in the Rwanda study. From all children with skin diseases, those with skin infections formed the largest part with percentages of 14.7 % and 17.6 % in the Ghanaian studies, 22.7 % in Rwanda and 27.7 % in Gabon. The highest prevalences were seen for tinea capitis and bacterial skin infections especially in the rural areas and schools with lower socioeconomic level.

Conclusions

The prevalences of skin diseases among African schoolchildren were high with a leading role for skin infections like tinea capitis and pyoderma.

Introduction

Several studies from African countries conducted over the past two decades have reported high prevalences of skin diseases among schoolchildren.[1-5] These skin diseases can affect the well-being and health conditions of the children.[6;7] To identify possible strategies for their prevention there is a great need to determine the burden of skin diseases in these communities.[1]

The majority of the data on the prevalence of skin diseases in Africa comes from hospital or dispensary-based records and does not necessarily represent the real prevalence of skin diseases within populations.[8] There are only few population-based studies on this subject.[8-12] The prevalence of one or more skin diseases among schoolchildren in Africa ranges between 35% and 80%.[5;9;11] The majority of the skin diseases found among schoolchildren are dominated by infections such as fungal infections and pyoderma.[5;10;13-15]

Elsewhere in the world similar patterns have been observed among schoolchildren in poor socioeconomic circumstances.[16-19] In industrialized countries, however, several hospital and population-based studies among schoolchildren showed much lower point-prevalences of fungal and other skin infections while the highest burden of skin diseases in these countries was formed by acne vulgaris and atopic dermatitis.[20-23]

The objective of this study was to determine the point-prevalences and the current spectrum of skin diseases among schoolchildren in rural and urban schools in three different African countries and to study the influence of the socioeconomic level (SEL).

Materials and Methods

Four cross-sectional studies with 4839 schoolchildren were conducted between 2004 and 2007. Specifically, these were carried out in Ghana (2004 and 2007), Gabon (2005) and Rwanda (2007). Details of the studies are presented in Table 1 and examples of some skin diseases are shown in Figure 1.

Ethical approval for the studies in Ghana was granted by the Institutional Review Board of the Noguchi Memorial Institute for Medical Research. The ethical approval number was CPN015 / 02-03. The study in Gabon was conducted with approval of the management of the Medical Research Unit of the Albert Schweitzer Hospital. In Rwanda the study was conducted in cooperation with a prevalence study of vernal keratoconjunctivitis in Rwandan schoolchildren and its association with atopy and parasitic infestation. Ethical approval was granted by the Rwandan National Ethics Committee.

The difference between skin diseases and skin disorders is not clear in the literature and is often subjective since both terms are used independently but are also often used in combination.[3;9;11;13;19] We defined skin diseases as an impairment of health or a condition of abnormal functioning of the skin, with fungal and bacterial skin infections, eczema

Table 1 Characteristics of the studies and baseline characteristics of the children in the different countries.*

	GHANA 2004	GHANA 2007	GABON	RWANDA
Region	Greater Accra Region: Accra Metropolitan Area and Ga West District	Greater Accra Region: Accra Metropolitan Area , Dangme East District and Ga East District	Albert Schweitzer Hospital which is located about 6 km from the city center of Lambaréné the capital of the Moyen-Ogooué province	Muhanga (Gitarama and Saki), Bugesera (Gicaca) and Kicuciro (Gicondo, Kigali)
Number of schools				
- Rural public (low SEL**)	2	6	1	3
- Urban public (low SEL)	1	3	1	3
- Urban private (middle SEL)	0	1	0	0
- Urban private (high SEL)	1	1	0	0
Part of these studies	Association of helminth infection with allergic sensitization and atopic eczema among schoolchildren. In cooperation with the department of Parasitology, Leiden University Medical Center.	EU project GLOFAL "Global view of food allergy: opportunities to study the influence of microbial exposure". In cooperation with the department of Parasitology, Leiden University Medical Center.	Association of helminth infection with allergic sensitization and atopic eczema among schoolchildren. In cooperation with the department of Parasitology, Leiden University Medical Center.	Prevalence of vernal keratoconjunctivitis in Rwandan schoolchildren and its association with atopy and parasitic infestation. In cooperation with the department of ophthalmology, Medical University Gent.
Number of children	463	1394	454	2528
Age distribution				
4-8	128 (27.6)	299 (21.4)	197 (43.4)	327 (12.9)***
9-12	275 (59.4)	804 (57.7)	188 (41.4)	1494 (59.1)
13-16	36 (7.8)	282 (20.2)	68 (15.0)	707 (28.0)
17-20	0	9 (0.6)	1 (0.2)	0
unknown	24 (5.2)	0	0	0
Sex				
Girls	201 (43.3)	734 (52.7)	227 (50.0)	1296 (51.3)
Boys	262 (56.6)	660 (47.3)	227 (50.0)	1224 (48.4)
Unknown	0	0	0	8 (0.3)
Characteristics of the schools				
Rural public (low SEL**)	226 (48.8)	753 (54.0)	209 (46.0)	1455 (57.6)
Urban public (low SEL)	125 (27.0)	214 (15.4)	245 (54.0)	1073 (42.4)
Urban private (middle SEL)	0	356 (25.5)	0	0
Urban private (high SEL)	112 (24.2)	71 (5.1)	0	0
Physical examination by dermatologist	A.A.H. , J.N.B.B., A.P.M.L	A.A.H., A.P.M.L.	A.A.H., A.P.M.L.	A.A.H.

* The contents of this table have been published before.[27]
** SEL: socioeconomic level.
***In Rwanda the youngest child was 8 years old.

Figure 1 Examples of children with dermatomycosis (**a**); tinea capitis (**b**); impetigo (**c**); eczema (**d** and **e**); and pityriasis rosea (**f**).

and psoriasis as the most important examples. In our study we considered skin disorders as skin diseases. Examples of skin disorders are acne vulgaris, benign nevi, freckles, hyperpigmentation, etc. Skin conditions were defined as symptoms or characteristics of the skin that were not considered as skin diseases or skin disorders. Examples of skin conditions are dry skin, keratosis pilaris, etc. All skin diseases found were subdivided into the following four categories: 1) skin infections (mycotic, bacterial, viral and parasitic), 2) inflammatory skin diseases, 3) benign skin tumors and nevi and 4) miscellaneous skin diseases. Skin conditions were categorized into a 5th category. The specific skin diseases and skin conditions which belong to these five categories are depicted in Table 2.

The presence of skin diseases and skin conditions was determined in all four studies by physical examination of all children by the same dermatologist (AAH) who was assisted by APML in both Ghanaian studies and Gabon and also by JNBB in the first Ghanaian study. The children were seen during a site-visit at school where the whole skin was inspected. The examination took place in a special room where privacy for each individual child was guaranteed. The skin findings were notified on a special intake form and in case of the presence of a skin disease photographs were taken of which some are shown in figure 1. The skin of each child was specifically examined for tinea capitis, pyoderma, inflammatory acne vulgaris, and eczema.[24-26] The reason to separate skin diseases and skin conditions as different entities is the possibility to compare the found prevalences of skin diseases with results of other epidemiological studies. In most epidemiological studies skin condition as depicted in category 5 are not evaluated and they increase overall prevalence rates. For the diagnosis of tinea capitis we looked for scaling on the scalp, hair loss, black dots, pustules and scars. We did not test for minimal infection, termed carrier state, i.e. we did not collect samples from all children. We, therefore, may have missed some children with asymptomatic dermatophyte scalp carriage so that the real prevalence of tinea capitis may even be higher.[24;26] Our clinical skills to diagnose tinea capitis were validated in the first Ghanaian study and in Gabon by direct microscopic examination and culture in the mycology laboratory of the department of Dermatology of the Leiden University Medical Centre (Leiden, the Netherlands).[10;24;26] The agreement between the clinical diagnosis and the results of microscopic examination as well as culture was high. In Ghana 31 (79.5%) of the 39 clinically suspected tinea capitis and in Gabon 74 (70.5%) of the 105 clinically suspected tinea capitis could be confirmed by KOH or culture. [24;26] We therefore did not collect hairs and skin scrapings in the Ghana 2007 study as well as in the Rwanda study and relied on our clinical diagnosis. In the current study, we only present the data of the clinical diagnosis of tinea capitis.

The diagnosis of pyoderma was used to describe any variant of superficial bacterial skin infection like impetigo, ecthyma, folliculitis, furuncle or tropical ulcer.[5] The Ghana 2004 study was performed in an area endemic for Buruli ulcer. The diagnosis of Buruli ulcer was made on clinical grounds and this disease was not seen in Gabon and Rwanda.

Table 2 Point-prevalences of skin diseases and skin conditions in African schoolchildren.

	Ghana 2004 N (%)	Ghana 2007 N (%)	Gabon 2005 N (%)	Rwanda 2007 N (%)
Number of children	**463**	**1394**	**454**	**2528**
One or more skin diseases (1-4)	**160 (34,6)**	**585 (42.0)**	**208 (45.8)**	**675 (26.7)**
One or more skin diseases and skin conditions total (1-5)	**206 (44.5)**	**642 (46.1)**	**220 (48.5)**	**736 (29.1)**
1. One or more skin infections total	**68 (14.7)**	**245 (17.6)**	**125 (27.7)**	**575 (22.7)**
One or more mycotic infections	**43 (9.3)**	**150 (10.8)**	**117 (25.8)**	**525 (20.8)**
Tinea capitis	39 (8.4)	121 (8.7)	105 (23.1)	522 (20.6)
Tinea other	4 (0.9)	8 (0.6)	12 (2.6)	3 (0.1)
Pityriasis versicolor	0 (0)	23 (1.6)	0 (0)	4 (0.2)
One or more bacterial infections	**28 (6.0)**	**95 (6.8)**	**8 (1.8)**	**33 (1.3)**
Pyoderma	20 (4.3)	81 (5.8)	7 (1.5)	32 (1.3)
Leg ulcers	1 (0.2)	11 (0.8)	2 (0.4)	0 (0)
Buruli ulcer	7 (1.5)	1 (0.1)	0 (0)	0 (0)
Rest bacterial	0 (0)	6 (0.4)	0 (0)	1 (0)
One or more viral infections	**4 (0.9)**	**10 (0.7)**	**6 (1.3)**	**25 (1.0)**
Verrucae	3 (0.6)	4 (0.3)	5 (1.1)	11 (0.4)
Mollusca contagiosa	1 (0.2)	0 (0)	0 (0)	9 (0.4)
Herpes simplex	0 (0)	6 (0.4)	1 (0.2)	1 (0.04)
Varicella	0 (0)	0 (0)	0 (0)	4 (0.2)
One or more parasitic infections	**0 (0)**	**1 (0.1)**	**0 (0.7)**	**1 (0.04)**
Scabies	0 (0)	1 (0.1)	3 (0.7)	1 (0.04)
2. One or more inflammatory skin diseases total	**38 (8.2)**	**148 (10.6)**	**51 (11.2)**	**109 (4.3)**
Acne vulgaris	15 (3.2)	66 (4.7)	5 (1.1)	33 (1.3)
Eczema	7 (1.5)	22 (1.6)	18 (4.0)	20 (0.8)
Seborrheic dermatitis	4 (0.9)	4 (0.3)	11 (2.4)	4 (0.2)
Prurigo simplex	9 (1.9)	52 (3.7)	17 (3.7)	52 (2.1)
Lichen simplex	1 (0.2)	4 (0.3)	2 (0.4)	0 (0)
Orthoergic eczema	0 (0)	2 (0.1)	0 (0)	0 (0)
Lichen planus	0 (0)	1 (0.1)	0 (0)	1 (0.04)
Psoriasis vulgaris	0 (0)	0 (0)	1 (0.2)	0 (0)
Pityriasis rosea	1 (0.2)	0 (0)	0 (0)	1 (0.04)
Alopecia areata	0 (0)	1 (0.1)	0 (0)	0 (0)
Granuloma annulare	1 (0.2)	0 (0)	0 (0)	0(0)

Table 2 Continued.

	Ghana 2004 N (%)	Ghana 2007 N (%)	Gabon 2005 N (%)	Rwanda 2007 N (%)
3. One or more benign skin tumors and nevi total	**4 (0.9)**	**61 (4.4)**	**2 (0.4)**	**3 (0.1)**
Normal nevi	0 (0)	51 (3.7)	0 (0)	0 (0)
Sebaceous nevus	0 (0)	1 (0.1)	0 (0)	0 (0)
Epidermal nevus	1 (0.2)	2 (0.1)	1 (0.2)	1 (0.04)
Café au lait macula	1 (0.2)	6 (0.4)	0 (0)	0 (0)
Depigmented nevus	2 (0.4)	0 (0)	0 (0)	0 (0)
Congenital nevus	0 (0)	0 (0)	0 (0)	1 (0.04)
Syringomata	0 (0)	1 (0.1)	0 (0)	0 (0)
Lipoma	0(0)	0 (0)	1 (0.2)	0 (0)
Granuloma pyogenicum	0 (0)	0 (0)	0 (0)	1 (0.04)
4. One or more miscellaneous skin diseases total	**78 (16.8)**	**225 (16.1)**	**63 (13.9)**	**3 (0.1)**
Traction alopecia	0 (0)	3 (0.2)	0 (0)	0 (0)
Miliaria/ heat rash	3 (0.6)	72 (5.2)	19 (4.2)	0 (0)
Papular urticaria	10 (2.2)	16 (1.1)	7 (1.5)	2 (0.1)
Scars	56 (12.1)	98 (7.0)	34 (7.5)	0 (0)
Keloids	3 (0.6)	12 (0.9)	0 (0)	0 (0)
Ulcers	0 (0)	1 (0.1)	0 (0)	0 (0)
Wounds	0 (0)	2 (0.1)	1 (0.2)	0 (0)
Striae	2 (0.4)	0 (0)	0(0)	0(0)
Albinism	0 (0)	2 (0.2)	0 (0)	0 (0)
Orange hair	0 (0)	1 (0.1)	0 (0)	0 (0)
Vitiligo	0 (0)	1 (0.1)	0(0)	0 (0)
Hyperpigmentation	0 (0)	6 (0.4)	3 (0.7)	1 (0.04)
Freckles	1 (0.2)	0 (0)	0 (0)	0 (0)
Varix	0 (0)	3 (0.2)	0 (0)	0 (0)
Nail problems	1 (0.2)	3 (0.2)	0 (0)	0 (0)
Acanthosis nigricans	0 (0)	3 (0.2)	0 (0)	0 (0)
Neurofibromatosis	1 (0.2)	0 (0)	0 (0)	0 (0)
Ichthyosis	2 (0.4)	2 (0.1)	0 (0)	0 (0)
Dysmorph syndrome	0 (0)	4 (0.3)	0 (0)	0 (0)
5. One or more skin conditions total				
Xerosis cutis	**83 (17.9)**	**110 (7.9)**	**35 (7.7)**	**85 (3.4)**
Keratosis pilaris	75 (16.2)	40 (2.9)	33 (7.3)	14 (0.6)
Pityriasis alba	7 (1.5)	43 (3.1)	2 (0.4)	75 (3.0)
Dyshidrosis lamellosis sicca	1 (0.2)	1 (0.1)	0 (0)	0 (0)
Fissure	0 (0)	26 (1.9)	0 (0)	0 (0)
Hyperkeratosis	0 (0)	0 (0)	1 (0.2)	0 (0)
	2 (0.4)	1 (0.1)	0 (0)	1 (0.04)

Cases of scabies were confirmed by direct microscopic examination with 20% potassium hydroxide (KOH) solution.

The diagnosis of inflammatory acne was defined by the presence of at least six pustules or papulopustules on the face.[25] The diagnosis of eczema was determined by the clinical diagnosis by a dermatologist.[27] Very dry skin among the children was diagnosed as xerosis cutis, a skin condition characterized by a dull color, rough texture and an elevated number of ridges.[28]

In Ghana, the rural and urban areas were selected according to the guidelines of the Ghana Statistical Service. The urban schools were all located in the Accra Metropolitan Area while the rural target areas were all settlements some distance from this area with low population densities and where the main income generating activities revolved around agriculture (fishing or farming). In Rwanda and Gabon the urban schools were located in the middle of the capital city or one of the major towns of the country, close to main roads while rural schools were situated in villages some distance from the main road with lower population densities than the urban areas.

Schools were selected for recruitment based on a broad social economic classification. This broad social economic classification reflects the average socioeconomic level (SEL), respectively low, middle and high. Schools located in these urban or rural areas were randomly selected from the district school lists, using a random digit generator sheet and according to probability proportional to size. Urban middle/high SEL schools were private fee-paying schools while urban low schools and rural schools were public, government-run schools where no school fees were paid.

Categorical data were analyzed for statistical differences by chi-square test. Point prevalences were estimated by calculating the proportions of children with skin diseases compared to the total groups of children with 95% confidence intervals. For the statistical analyses, we used SPSS for Windows version 17.0 (SPSS Inc, Chicago, IL, USA) and software which is freely available on the internet (http://www.dimensionresearch.com/resources/calculators/conf_prop.html).

Results

The characteristics of the studies and the baseline characteristics of the children in the different countries are presented in Table 1 and have been previously published.[27] In the two Ghanaian studies out of 15 schools screened 8 were rural schools, 4 were urban public schools and 3 were schools with middle to high SEL. In Gabon and in Rwanda only schools with a low SEL were screened, equally divided between rural and urban. Most children were between 4 and 16 years old and boys and girls were equally distributed (Table 1).

The point-prevalences of skin diseases and skin conditions among the schoolchildren are presented in Table 2. The prevalences of children with one or more skin diseases (category 1-4) were high; 34.6% and 42% in the Ghanaian studies, 45.8% in Gabon and 26.7%% in the Rwanda study.

Skin infections formed the largest group of all skin diseases with percentages of 14.7% and 17.6% in the Ghanaian studies, 27.7% in Gabon and 22.7% in Rwanda which corresponds with percentages of 42.5%, 41.9%, 60.1% and 85.2% relative to all skin diseases in the four studies respectively. Within the group of skin infections, the highest prevalences were seen for tinea capitis (8.4% in the Ghana 2004 study, 8.7% in the Ghana 2007 study, 20.6% in the Rwanda study and 23.1% in the Gabon study).

Relatively high prevalences were also found for bacterial skin infections with prevalences ranging between 1.3% in the Rwanda study and 6.8% in the Ghana 2007 study. The prevalences found for scabies and viral infections like verrucae vulgaris and mollusca contagiosa were very low. Buruli ulcer was only found in Ghana.

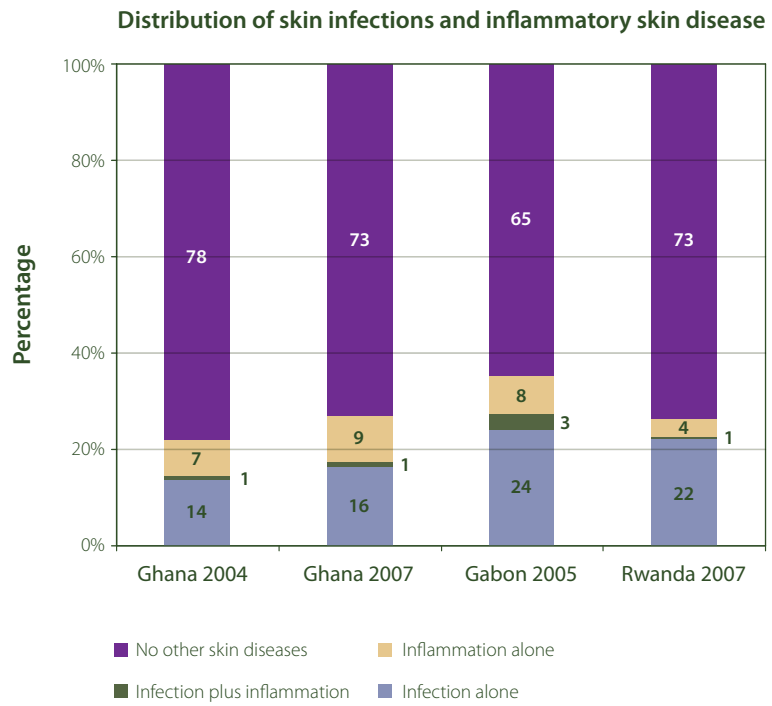
The point-prevalences of eczema ranged between 0.8% in Rwanda and 4.0% in Gabon and have been published before.[27] Acne vulgaris showed prevalences of 3.2 % and 4.7% in the studies performed in Ghana while these percentages were lower with 1.1% in Gabon and 1.3% in Rwanda. The prevalences of other inflammatory skin diseases like lichen planus, psoriasis and CDLE were low in our study.[20;21;23;29]

Heat rash (miliaria) was most frequently seen in the Ghana 2007 and Gabon studies with percentages of 5.2% and 4.2%. With regards to skin conditions (Table 2, category 5) our studies showed much higher prevalences of xerosis cutis in Ghana 2004 and Gabon compared with Ghana 2007 and Rwanda.

In other studies skin infections and inflammatory skin diseases are the most prevalent and also have the most impact on the children's health and, therefore, we present these categories separately in figure 2 to allow a better comparison with other past and future studies. The combined prevalences of infectious and inflammatory skin diseases (category 1 and 2) were 22% in the Ghana 2004, 26% in the Ghana 2007, 35 % in the Gabon and 27% in the Rwanda study.

The prevalences of the 8 most frequent skin diseases and skin conditions are presented separately for the urban and rural schools in Table 3. In all 4 studies the prevalences of tinea capitis were higher in the rural schools (Table 3). The same pattern was seen for pyoderma with higher prevalences in the rural school with the exception of the Gabon study where a higher prevalence of pyoderma was found in the urban school (Table 3). Acne vulgaris was more frequently seen in the urban schools (Table 3). Especially in the Ghana 2007 study there was an important difference between the urban and rural schools (10.1% versus 0.1%) because in this study two urban schools with a higher/middle SEL were included where acne vulgaris was more prevalent.[25] Prurigo simplex and xerosis cutis were most frequently seen in the rural areas (Table 3).

Figure 2 The distribution of skin infections and inflammatory skin diseases.



In Table 4 the differences in prevalences of the 8 most frequent skin diseases and skin conditions are shown according to the SEL. Because the schools in Gabon and Rwanda were all of low SEL, only the Ghana studies are shown. In the schools with a low SEL tinea capitis was seen much more frequently (with percentages of 10.8% and 10.3%), compared to the prevalences in schools with a middle/high SEL (with prevalences of 0.9% and 4.9%). Considerable differences were also seen for pyoderma where we saw the same pattern; high prevalences of 5.1% and 7.0% in schools with a low SEL and lower prevalences of 1.8% and 3.0% in schools with a middle/high SEL. Among schoolchildren with a middle/high SEL the prevalences of acne vulgaris and eczema were significantly higher comparing the schoolchildren with a low SEL as has been published before.[25;27] Other significant differences were seen for xerosis cutis. Higher prevalence rates were found among schoolchildren with a low SEL (19.7% and 4%) compared with schoolchildren with a middle/high SEL (5.4% and 0.2%).

Table 3 Prevalences of the most common skin diseases and skin conditions in rural and urban schools.*

	GHANA 2004 N (%) Total 463			GHANA 2007 N (%) Total 1394			GABON N (%) Total 454			RWANDA N (%) Total 2528		
Urban / Rural N (%)	Urban n/ N (%) (95%CI)	Rural n/N (%) (95%CI)		Urban n/N (%) (95%CI)	Rural n/N (%) (95%CI)		Urban n/N (%) (95%CI)	Rural n/N (%) (95%CI)		Urban n/N (%) (95%CI)	Rural n/N (%) (95%CI)	
Number of children	237	226		641	753		245	209		1073	1455	
Tinea capitis	9 (3.8) (1.4;6.2)	30 (13.3) (2.7;8.8)		42 (6.6) (4.7;8.5)	79 (10.5) (8.3;12.7)		50 (20.4) (15.4;25.5)	55 (26.3) (20.4;32.3)		204 (19.0) (16.7;21.4)	318 (21.9) ((19.7;24.0)	
Pyoderma	10 (4.2) (1.7;6.8)	10 (4.4) (1.7;7.1)		*21 (3.3) (1.9;4.7)*	*60 (8.0) (6.0;9.9)*		4 (1.6) (0.1;3.2)	3 (1.4) (0.0;3.1)		11 (1.0) (0.4;1.6)	21 (1.4) (0.8;2.1)	
Acne vulgaris	7 (3.0) (0.8;5.1)	8 (3.5) (1.1;6.0)		***65 (10.1)*** *(7.8;12.5)*	*1 (0.1) (0.0;0.4)*		3 (1.2) (0.0;2.6)	2 (1.0) (0.0;2.3)		18 (1.7) (0.9;2.5)	15 (1.0) (0.5;1.6)	
Eczema	5 (2.1) (0.3;3.9)	2 (0.9) (0.0;2.1)		12 (1.9) (0.8;2.9)	10 (1.3) (0.5;2.2)		6 (2.4) (0.5;4.4)	12 (5.7) (2.6;8.9)		10 (0.9) (0.4;1.5)	10 (0.7) (0.3;1.1)	
Prurigo simplex	0 (0)	9 (4.0) (1.4;6.5)		16 (2.5) (1.3;3.7)	36 (4.8) (3.3;6.3)		*1 (0.4) (0.0;1.2)*	*16 (7.7) (4.1;11.3)*		*12 (1.1) (0.5;1.8)*	*40 (2.7) (1.9;3.6)*	
Papular urticaria	4 (1.7) (0.1;3.3)	6 (2.7) (0.6;4.8)		7 (1.1) (0.3;1.9)	9 (1.2) (0.4;2.0)		6 (2.4) (0.5;4.4)	1(0.5) (0.0;1.4)		1 (0.1) (0.0;0.3)	1 (0.1) (0.0;0.2)	
Xerosis cutis	*27 (11.4) (7.4;15.4)*	*48 (21.2) (15.9;26.6)*		*2 (0.3) (0.0;0.7)*	*38 (5.0) (3.5;6.6)*		*5 (2.0) (0.3;3.8)*	*28 (13.4) (8.8;18.0)*		3 (0.3) (0.0;0.6)	11 (0.8) (0.3;1.2)	
Keratosis pilaris	3 (1.3) (0.0;2.7)	4 (1.8) (0.1;3.5)		27 (4.2) (2.7;5.7)	16 (2.1) (1.1;3.2)		1 (0.4) (0.0;1.2)	1 (0.5) (0.0;1.4)		31 (2.9) (1.9;3.9)	44 (3.0) (2.1;3.9)	

*Italic is statistically significant

Table 4 Differences in prevalences of the most common skin diseases and conditions between low and middle/high SEL in Ghana.

	GHANA 2004 n/N (%) (95%CI) Total 463		GHANA 2007 n/N (%) (95%CI) Total 1394	
Socioeconomic (SEL)	Low N (%)	Middle/High N (%)	Low N (%)	Middle/High N (%)
Number of children	351	112	967	427
Tinea capitis	*38 (10.8) (7.6;14.1)**	*1(0.9) (0.9;2.6)**	*100 (10.3) (8.4;12.3)**	*21 (4.9) (2.9;7.0)**
Pyoderma	18 (5.1) (2.8;7.4)	2 (1.8) (0.7;4.2)	*68 (7.0) (5.4;8.6)**	*13 (3.0) (1.4;4.7)**
Acne vulgaris	11 (3.1) (1.3;5)	4 (3.6) (0.1;7.0)	*19 (2.0) (1.1;2.8)**	*47 (11.0) (8.0;14.0)**
Eczema	2 (0.6) (0.2;1.4)	5 (4.5) (0.6;8.3)	12 (1.2) (0.5;1.9)	10 (2.3)(0.9;3.8)
Prurigo simplex	9 (2.6) (0.9;4.2)	0 (0.0)	38 (3.9) (2.7;5.2)	14 (3.3) (1.6;5.0)
Papular urticaria	6 (1.7) (0.4;3.1)	4 (3.6) (0.1;7.0)	10 (1.0) (0.4;1.7)	6 (1.4) (0.3;2.5)
Xerosis cutis	*69 (19.7) (15.5;23.8)**	*6 (5.4) (1.2;9.5)**	*39 (4.0) (2.8;5.3)**	*1 (0.2) (0.0;0.7)**
Keratosis pilaris	5 (1.4) (0.2;2.7)	2 (1.8) (0.7;4.2)	26 (2.7) (1.7;3.7)	17 (4.0) (2.1;5.8)

*Italic is statistically significant

Discussion

The prevalences of skin diseases among schoolchildren in Ghana, Gabon and Rwanda varied between 27% and 46%. Focusing on only skin infections and inflammatory skin diseases these prevalences were 22% and 27% in the two Ghanaian studies and 35% in Gabon and 27% in Rwanda, respectively.

Of all skin diseases, skin infections were the most prominent cause which confirms earlier studies in African children.[5;9-11] Factors such as overcrowding, malnutrition and climatic conditions such as heat and humidity can lead to an increase in fungal and bacterial infections in tropical and semi-tropical countries.[3;4;9;12;30]

Tinea capitis was the most prevalent skin infection in all four studies. With a prevalence of 9% the Ghana 2007 study matched the prevalence of 8% found in the Ghana 2004 study.[24] The higher prevalence of 21% in the Rwanda study resembled the prevalence of 26% found in the Gabon study.[26] The difference between the prevalences in the Gabon and Rwanda study and both Ghanaian studies is remarkable. One of the reasons for this

difference may be that both Ghanaian studies also included schools with a high or middle SEL where the prevalences of tinea capitis were much lower. Additionally, in most schoolchildren in Ghana the scalps were shaven, reducing the contagiousness, while in Gabon and Rwanda girls wore longer hairstyles. The Ghana Education Service requires that boys and girls in basic and secondary public schools cut their hair very short. It seems that this rule is actually beneficial.[31] However there is also evidence that in some cases the use from a common source (e.g. haircutting or barber) and the cutting of the hair itself may introduce an infection. In all four studies tinea capitis was more prominent in rural areas and in schools with a low SEL, but the differences in Gabon and Rwanda were much smaller. This may be explained by the fact that the differences between rural and urban areas in Rwanda and Gabon were less pronounced than those in Ghana and the fact that only schools with a lower SEL were studied in these countries. Tinea capitis is highly contagious especially at family level. A low SEL and consequent overcrowding appears to be a major risk factor for tinea capitis.[5;9;11;16] Although the clinical appearance is variable, late detection and lack of treatment of this disease can result in widespread infections and, in rare cases, permanent alopecia.[32] Tinea capitis is spread worldwide and a major public health concern.[33]

With prevalences of 4% and 6% in Ghana and 2% and 1% in Gabon and Rwanda, pyoderma was the second highest cause of skin infection in our study. The climate in Rwanda is much cooler and the humidity lower which can explain the lower prevalence of bacterial skin infections in this country compared with Ghana. For the lower prevalence of bacterial skin infections in Gabon we do not have a good explanation. Other point-prevalence studies among schoolchildren in Ethiopia, Mali and 2 studies in Tanzania showed prevalences of pyoderma comparable with our Ghana studies, while another study from Kenya showed a higher prevalence of 12.7%.[8;10;13;34;35] In the study from Kenya the prevalence of bacterial infections was even slightly higher than fungal infections.[35]

The low prevalence of scabies in our study is remarkable but not exceptional. Prevalence rates ranging from 0.7% to 30.4% in other studies document considerable regional differences, probably due to differences in socioeconomic situations in the various countries.[11;34-37] In some studies high prevalences of parasitic infestations like scabies are seen.[9-11;13;38] Despite thorough inspection of the scalp we didn't find any case of pediculosis capitis. Although this disease is endemic among Caucasian children in Europe and Northern America, it was not observed in studies from west Africa, maybe due to the different hair type.[11;34;39;40] However in studies from east Africa and especially Ethiopia prevalences ranging from 3.6 % to 57.1% are mentioned.[8;9;41]

The prevalence of mollusca contagiosa was low; most probably because most children who were examined were above the age of 8 years (see Table 1). The low prevalence rate found for verrucae vulgaris was comparable with most other community based studies among schoolchildren in sub Sahara Africa.[9;10;13;35]

With the exception of the Buruli ulcer in Ghana, like in other studies, the classical tropical diseases such as leprosy or filarial lymph edema were not found among schoolchildren in this study. The exotic aspect of tropical dermatology seems to be over-emphasized in the literature.[5;13;34;35]

The prevalences of acne vulgaris and eczema were higher in the urban areas and in both Ghanaian studies especially in the schools with a middle/high SES, but were still lower in comparison with industrialized countries.[25;42] With the arrival of urbanization and westernization in developing countries, it is believed that the prevalence of acne vulgaris and eczema will increase to the level of industrialized countries.

The prevalence of heat rash (miliaria) was higher in the Ghana 2007 and Gabon studies which could be explained by differences in temperature and humidity in the different countries and in different time periods. The Ghana 2007 study was performed in March which is normally the end of the dry season with high temperatures. The Ghana 2004 study was performed in June which is in the rainy season when the temperatures are somewhat lower. Gabon has a typical tropical climate with high temperatures and high humidity. However Gabon also has a dry and wet season similar to Ghana. The temperature and humidity in the mountainous Rwanda are much lower.

In the four studies xerosis cutis was seen most frequently in the rural areas with the highest prevalences in the Ghana 2004 and the Gabon study. The etiology of xerosis cutis is multifactorial. The role of the barrier function of the stratum corneum is important. When the barrier is impaired the skin will be dry because of trans-epidermal water loss and will be more vulnerable for both infectious and inflammatory skin diseases.[43;44] Frequent washing with soap is considered to be a healthy exercise in Ghana and can damage the barrier function of the skin. Despite the advice to use less soap, children often continue their habits, also due to the hot, humid climate and the subsequent sweating which may lead to dry skin.[42] Differences between temperature and humidity could partly explain the different prevalences of xerosis cutis in the four studies. Prurigo simplex, like xerosis cutis, had the highest prevalences in the rural areas suggesting a relationship between these two conditions. It is plausible that xerosis cutis could lead to prurigo simplex.

The strength of our study is that we studied large numbers of schoolchildren in three different countries and that all children were seen by a dermatologist or a team of dermatologists. A limitation of our study is that when one measures a point-prevalence there is always a chance of underestimation especially in diseases with a chronic relapsing course.

While there are certainly some advantages by differentiating skin diseases from skin conditions, this may also lead to some difficulties and limitations. The main problem is that most investigators in the past did not make this difference so that their data cannot be directly compared with the data of the current study. On the other hand, future investigators could use the definitions used in the current study as a guidance to design their new study, so that more precise prevalence data of the different skin diseases and skin conditions could be obtained.

Most skin diseases found in our and other studies from developing countries were not life threatening and could easily be treated.[8;35;45-47] However, they can potentially harm the skin, the health and well-being of children affected. Skin diseases are often not considered of high priority as was concluded in a recent WHO discussion paper.[5] The occurrence of skin diseases does not only depend on improved treatment schemes within the health system but also on improvement of socioeconomic conditions.[45;48] In addition to the need to improve the quality and availability of skin disease treatment in Africa, there is also a great need to determine the burden of skin diseases in communities and to identify possible strategies for their prevention.[1]

Acknowledgements

The authors thank I. Larbi, Y. Aryeetey and B. Obeng for their support.

The study was made possible by a gift from the Gratama Foundation, the Netherlands, by EU-project GLOFAL 'Global view of food allergy: opportunities to study the influence of microbial exposure' FP6-2003-Food-2B contract: FOOD-CT-2005-517812, by The Netherland Organization for Scientific Research for Global Development, WOTRO grant number WB 93-433 and by the cooperation with the University of Gent, Belgium.

We are grateful to all children, parents and teachers for their cooperation and like to thank the fieldworkers for their participation and enthusiasm

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Chapter 3

Prevalence of symptomatic tinea capitis and associated causative organisms in the Greater Accra Region, Ghana

British Journal of Dermatology 2006 **154**, pp 784-786
Published as Corresponding letter

Hogewoning AA [1], Duijvestein M [2], Boakye D [3], Amoah AS [3],
Obeng BB [3], van der Raaij-Helmer EMH [4], Staats CCG [4], Bouwes Bavinck JN [4],
Yazdanbakhsh M [2], Lavrijsen APM [4]

[1] Dermatology, University of Ghana Medical School, Korle-Bu Teaching Hospital, Accra, Ghana,
[2] Parasitology, Leiden University Medical Centre, Leiden, The Netherlands,
[3] Noguchi Memorial Institute for Medical Research, University of Ghana, Legon, Ghana,
[4] Dermatology, Leiden University Medical Centre, Leiden, The Netherlands

Abstract

Background

Infections with tinea capitis are endemic among school children in Africa. The objectives of this study were to determine the prevalence and causative species of tinea capitis among school children in different rural and urban schools in Ghana.

Methods

A cross-sectional study was performed from may 31st to June 6th 2004 with 475 children aged between 5-17 years, and residing in two rural and two urban schools in the Greater Accra Region (Ghana). All children were clinically examined for tinea capitis. Symptomatic children were tested by microscopic examination using 20% potassium hydroxide solution (KOH) and cultivation on Sabouraud's dextrose agar with chloramphenicol.

Results

Based on clinical examination, 39 (8,4%) out of 463 children had tinea capitis. A total of 31 (6.7%) children were positive by direct examination (KOH) and/or fungal culture. The prevalence of tinea capitis largely depended on the school studied, ranging from 0.9% in an urban school with a high socioeconomic status to more than 10% in the two rural schools with low socioeconomic status. Similarly, the spectrum of causative species varied importantly between the different schools. Taken all schools together, T. violaceum (25.9%) was the most prominent species, followed by T. tonsurans (22.2%) and M. audouinii (14.9%).

Conclusions

Tinea capitis is endemic among school children in the Greater Accra Region (Ghana). The prevalence of tinea capitis and the causative species largely depended on the type of school that was investigated.

Introduction

Tinea capitis is a common infection among school children throughout the world.[1] It is a superficial fungal infection of the scalp, which may be caused by Trichophyton and Microsporum species. The most important causative agents are species which are causing an endothrix infection, such as T. gourvilli, T. soudanense, T. tonsurans, T. violaceum and T. yaoundei and species that cause an ectothrix infection such as M. audouinii, M. canis and M. gypseum.[2] The causative agent of tinea capitis varies with geography, socioeconomic status and time.[3]

Though the clinical appearance is variable, late detection and lack of treatment of this disease can result in widespread infections and, in rare cases, permanent alopecia.[4] More knowledge about the prevalence and causative agents of tinea capitis is necessary to improve control and therapeutic measures. Several investigations about this public health problem have been published but to our knowledge no research has been carried out in Ghana, recently.

In order to provide information about this disease in the country, a cross-sectional study was conducted in the Greater Accra Region, comparing two rural schools with two urban schools. One urban school had a high, the three others schools a low socioeconomic status. The study was designed a) to access the prevalence of tinea capitis in the different schools, and b) to identify the causative agents.

Patients and Methods

Study site

Accra is the capital city of Ghana, situated about 25 kilometres west of the Greenwich Meridian. Ghana lies almost in the middle of West Africa and has a typical tropical climate, with average temperatures between 25.5°C and 27°C. The humidity is constantly high, at about 80%.

Study population and collection of material

From May 31st to June 6th 2004, 463 school children from two public rural schools, one public urban school and one private urban school in the Greater Accra Region were fully clinically examined for skin diseases by a team of three dermatologists (Figure 1).

Socioeconomic status was determined by questionnaires on parameters such as occupation of both parents and the educational background of the responding parent. By this method, the urban private school (University Primary) had been identified as a school with high socioeconomic status and the public urban school (James Town) and two rural schools (Mayera and Ayikai Doblo) had been classified as schools with low socioeconomic status.

Figure 1 Photographs of the four participating schools. Clockwise starting in the upper left corner: James Town (JT), University Primary (UP), Aykai Doblo (AD) and Mayera (MA).

Specific attention was focused on clinical signs of fungal infection on the scalp (scaling, hair loss, black dots, pustules and scars). Scalp samples were taken by gentle brushing with glass slides and tweezers, and transported at room temperature to the Mycology Laboratory of the Department of Dermatology of the Leiden University Medical Centre in Leiden, The Netherlands.

Laboratory methods

The specimens were examined by direct microscopic examination using 20% potassium hydroxide solution (KOH). For the cultures modified Sabouraud's dextrose agar with chloramphenicol was used and the specimens were incubated for 28 days at a temperature of 28º degrees Celsius. Species identification was based on growth rate, macroscopic aspect and microscopic examination. Four samples that could not be identified were sent to the National Institute of Public Health and the Environment (RIVM), Bilthoven, the Netherlands for characterisation.

Statistical analysis

Chi-square analyses were used to analyze the different distribution of tinea capitis among boys and girls, different age groups and the different schools.

Results

Figure 2 shows examples of clinical fungal infection on the scalp in the study population. Of the 463 children examined during the survey, a total of 39 (8.4%) had clinical

Figure 2 Tinea Capitis caused by T. Violaceum in a 12-year old boy (**a**) and a 6-year old girl (**b**). Gray patches caused by M. Audouinii in a 5-year old girl (**c**) and a 8-year old boy (d).

signs of tinea capitis (Table 1). In 4 out of the 39 clinically suspected cases no sufficient material could be collected for microscopic examination and culture. Of the remaining 35 patients, hair stumps and scales were collected and 27 were positive during microscopic examination and 27 cases were identified by culture (Table 1).

Table 1 Demographic data for tinea capitis among school children in Greater Accra Region, Ghana.

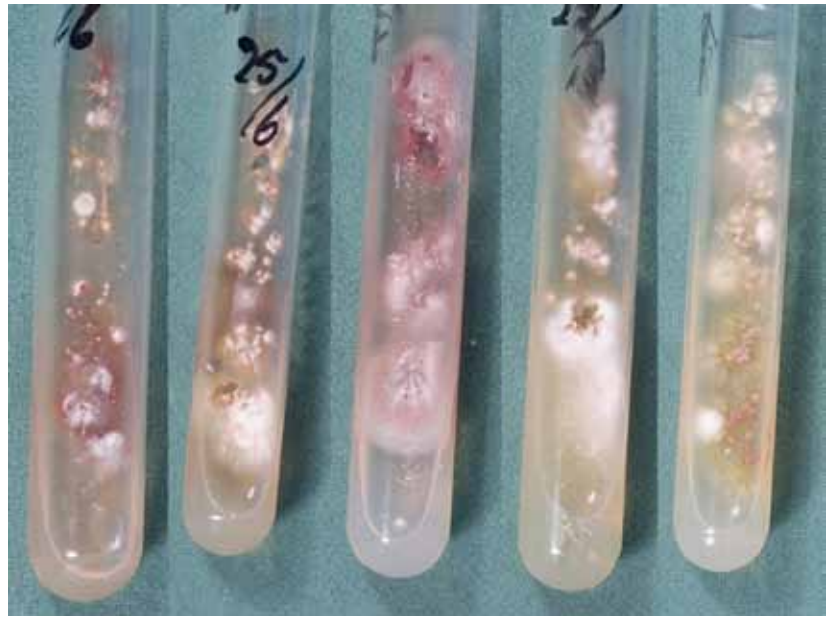
	N	Positive by physical examination N (%)	Positive by KOH, only N (%)	Positive by Culture, only N (%)	Positive by KOH and/or culture N (%)
All children together	463	39 (8.4)	27 (5.8)	27 (5.8)	31 (6.7)
Gender					
Male	262	26 (9.9)	17 (6.5)	16 (6.1)	20 (7.6)
Female	201	13 (6.5)	10 (5.0)	11 (5.5)	11 (5.5)
Age (years)					
5-8	130	10 (7.7)	7 (5.4)	7 (5.4)	8 (6.2)
9-11	196	17 (8.7)	11 (5.6)	13 (6.6)	13 (6.6)
12-17	104	6 (5.8)	5 (4.8)	5 (4.8)	6 (5.8)
Age not known	33				
Schools*					
Mayera (rural, low SES)	123	15 (12.2)	9 (7.3)	11 (8.9)	12 (9.8)
Ayikai Doblo (rural, low SES)	103	15 (14.6)	10 (9.7)	8 (7.8)	11 (10.7)
James Town (urban, low SES)	125	8 (6.4)	7 (5.6)	7 (5.6)	7 (5.6)
Univ. Prim. (urban, high SES)	111	1 (0.9)	1 (0.9)	1 (0.9)	1 (0.9)

*SES = socioeconomic status; Univ. Prim. = University Primary

Figure 3 shows representative cultures of different species from the study group. Of the 35 samples, a total of 23 samples were positive for both microscopic examination and culture, 4 were positive for microscopic examination and negative for culture, 4 were negative for microscopic examination and positive for culture and 4 were negative for microscopic examination and culture.

Tinea capitis was slightly more prevalent among boys compared to girls and in the age group of 5 to 11 years compared to the 12 to 17 year age group but these variations were

Figure 3 Colonies of T. violaceum, T. tonsurans, T. rubrum, T. ferrugineum and T. concentricum (from left to right).



not statistically significant (Table 1). Tinea capitis was, however, significantly more prevalent in the rural schools (more than 10%) compared to the urban school with a high socioeconomic status (University Primary) ($p < 0.001$) (Table 1). The urban school with a low socioeconomic status (James Town) occupied an intermediate position (about 6%) regarding the prevalence of tinea capitis (Table 1).

The frequency distribution of the different species among the different schools is depicted in Table 1. Combining the data of the schools together, the most frequently detected species were T. violaceum (25.9%), T. tonsurans (22.2%) and M. audouinii (14.9%). The frequency distribution of the different species was quite different between the different schools. Infection with T. tonsurans accounted for half of the patients in the Mayera school and for the single case in the University Primary school, whereas infections with T. rubrum and T. concentricum were only present in James Town, and with T. ferrugineum only in the Ayikai Doblo school (Table 2). Infections with T. violaceum, M. audouinii, and F. solani were found in different schools.

Table 2 Results of the 35 cultures according to the different schools.

	Schools**				Distribution	
Isolated species*	**MA**	**AD**	**JT**	**UP**	**Total**	**(%)*** **
Total number of cultures	13	12	9	1	35	
- Isolation of fungal species	10	8	7	1	27	
T. violaceum	3	3	1	0	7	25.9
T. tonsurans	5	0	0	1	6	22.2
M. audouinii	2	2	0	0	4	14.9
T. rubrum	0	0	3	0	3	11.1
T. concentricum	0	0	2	0	2	7.4
T. ferrugineum	0	2	0	0	2	7.4
F. solani	0	1	1	0	2	7.4
M. gypseum	1	0	0	0	1	3.7
- Results of other cultures						
Saprophyte	1	2	0	0	3	
Bacteria	1	0	0	0	1	
Sterile	0	2	2	0	4	

*T = Trichophyton; M = Microsporum; F = Fusarium.
**MA = Mayera; AD = Ayikai Doblo; JT = James Town; UP = University Primary school
***Frequency distribution among the 27 cultures with isolation of fungal species.

Discussion

Our survey showed that tinea capitis is endemic among school children in the Greater Accra Region, Ghana. The overall prevalence of tinea capitis was 8.4% and varied from 1% in the urban school with a high socioeconomic status to more than 10% in the rural schools with low socioeconomic status. The higher prevalence of tinea capitis in the rural schools with a low socioeconomic status might be explained by a lower availability of antifungal treatments, poor hygienic conditions, school and household overcrowding. The prevalence of tinea capitis was highest among boys in the younger age group (5-11 years) as reported in earlier studies,[5-7] although in our study statistical significance was not reached. A possible shortcoming of the study is that we did not test for sub-clinical infection, i.e. we did not collect hairs from children without active

disease. We, therefore, may have missed some children with sub-clinical infection so that the real prevalence of tinea capitis may even be higher.

Taken all schools together, *T. violaceum* (25.9%) was the most prominent species in our study followed by *T. tonsurans* (22.2%). The percentage of *T. violaceum* was lower than recent figures found in South Africa (90%),[8] and Rwanda (42%).[9] The percentage of *M. audouinii* (14.8%) was relatively low in comparison to other studies performed in Africa.[5;9-11] We found *T. rubrum*, the most common dermatophyte worldwide but not a frequently reported cause for tinea capitis, in a percentage of 11.1%. No *T. soudanense* was found, while in other studies performed in the region this species showed to be one of the most frequent causative agents.[5;7;12]

The prevalence of tinea capitis as well as the spectrum of fungal species varied considerably between the schools. Thus it should be noted that prevalence data of studies performed in one area of the country cannot be generalized as epidemiological data for the entire country.

We did not find any child with extensive disabling scarring tinea capitis. A considerable number of children, however, showed some minor scars on the scalp without evidence of active infection. Apparently, most children with tinea capitis underwent spontaneous recovery without any treatment resulting in no or only minor scarring.

In summary, our study showed that tinea capitis is endemic among school children in the Greater Accra Region in Ghana and that the prevalence largely depended on the location and the socio-economic status of the school that was investigated. Additionally, there were important varieties in the causative species described between the different schools in our study group and the causative species were also different compared to studies conducted in other African countries. [13;14] [15]

Acknowledgements

The authors acknowledge Irene Akosua Larbi, who helped with recruiting school children, the Director of the Institute, Prof. D. Ofori-Adjei for permission to have the study done at the Institute, and Prof. M.D. Wilson for support.

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Chapter 4

Prevalence and causative fungal species of tinea capitis among schoolchildren in Gabon

Mycoses 54 (5): E354-E359 Sep 2011

Hogewoning AA [1,2,3], Adegnika AA [4,5,6], Bouwes Bavinck JN [3],
Yazdanbakhsh M [5], Kremsner PG [4,6], van der Raaij-Helmer EMH [3],
Staats CCG [3],Willemze R [3], Lavrijsen APM [3]

[1] Department of Dermatology, University of Ghana Medical School, Korle-Bu Teaching Hospital, Accra, Ghana,
[2] Department of Dermatology, King Faisal Hospital, Kigali, Rwanda,
[3] Department of Dermatology, Leiden University Medical Centre, Leiden, The Netherlands,
[4] Albert Schweitzer Hospital Lambarene, Gabon,
[5] Department of Parasitology, Leiden University Medical Centre, Leiden, The Netherlands.
[6] Institute of Tropical Medicine, University of Tübingen, Tübingen Germany

Summary

Tinea capitis is endemic among schoolchildren in tropical Africa. The objective was to determine the prevalence of symptomatic tinea capitis in schoolchildren in Gabon. A cross-sectional study was conducted with 454 children aged 4 to 17 years, and attending a rural and an urban school. The diagnosis of tinea capitis was based on clinically manifest infection, direct microscopic examination using 20% potassium hydroxide (KOH) solution and fungal culture. Based on clinical examination, 105 (23.1%) out of 454 children had tinea capitis. Seventy-four (16.3%) children were positive by direct examination (KOH) and/or fungal culture. The prevalence of tinea capitis depended on the school studied and ranged from 20.4 % in the urban school with a higher socioeconomic status to 26.3 % in the rural school with lower socioeconomic status. Similarly, the spectrum of causative species varied between the different schools. Taken the schools together, *T. soudanense* (29.4%) was the most prominent species, followed by *T. tonsurans* (27.9%) and *M. audouinii* (25.0%). Clinically manifest tinea capitis is endemic among schoolchildren in the Lambaréné region in Gabon. The prevalence of tinea capitis and the causative species depended on the type of school that was investigated.

Introduction

Superficial fungal infections of the scalp (tinea capitis) are endemic among schoolchildren especially in tropical Africa and they can cause significant public health problems.[1] Tinea capitis is caused by *Trichophyton* and *Microsporum* species.[2] The most important causative agents are species which are causing an endothrix infection, such as *T. gourvilli*, *T. soudanense, T. tonsurans*, *T. violaceum* and *T. yaoundei* and species that cause an ectothrix infection such as *M. audouinii*, *M. canis* and *M. gypseum*.[3] The causative agent of tinea capitis varies with geography, socioeconomic status and time.[4]

Although the clinical appearance is variable, late detection and lack of treatment of this disease can result in widespread infections and, in rare cases, permanent alopecia.[5] More knowledge about the prevalence and causative agents of tinea capitis is necessary to improve control and therapeutic measures.

The objectives of this study were to summarize previous prevalence studies in sub-Saharan African countries and to determine the prevalence of tinea capitis and identify the causative species among schoolchildren in a rural and urban school in Gabon.

Materials and Methods

To collect information about tinea capitis in Gabon, a cross-sectional study was conducted in the Lambaréné Region, comparing a rural school with an urban school (Figure1). The rural school (Zile school) had a low and the urban school (Lalala school) had a higher socioeconomic status.

The study was carried out from the Albert Schweitzer Hospital which is located about 6 km from the city centre of Lambaréné, Gabon. This city is situated at the riverside of the Ogooué River – one of the largest rivers in Central Africa. Gabon is sparsely populated with an estimated total population of around 1.3 million people in a country of 267.667 km^2. Most of the territory is covered by dense tropical rainforest. The country has a typical tropical climate with two rainy and two dry seasons and average temperatures between 25.5°C and 27°C. The humidity is constantly high, at about 80%.

In January 2005, 454 schoolchildren from the two schools were fully clinically examined for skin diseases by two dermatologists (A.A.H., A.P.M.L.). Specific attention was focused on clinical signs of fungal infection on the scalp (scaling, hair loss, black dots, chicken skin effect, pustules and scarring alopecia). Samples from scales and hairs were taken by gentle brushing with glass slides and tweezers (Figure 1), and transported at room temperature to the Mycology Laboratory of the Department of Dermatology of the Leiden University Medical Centre in Leiden, the Netherlands. The samples can be kept at room temperature for several months (and probably longer), without affecting the viability of the fungi.[6]

Figure 1 Panel (**a**) shows schoolchildren in the urban school (Lalala) and panel (**b**) in the rural school (Zile). Panel (**c**) shows gentle brushing with glass slides and panel (**d**) collection of scales with tweezers. Panel (**e**) shows a non-inflammatory type of tinea capitis with 'gray-patch' scaling caused by M. *audouinii* in a 6-year old boy and panel (**f**) shows tinea capitis caused by T. *soudanense* in a 4-year old boy.

The specimens were examined by direct microscopic examination using 20% potassium hydroxide solution (KOH). For the cultures modified Sabouraud's dextrose agar with chloramphenicol was used and the specimens were incubated for 28 days at a temperature of 28º degrees Celsius. Species identification was based on growth rate, macroscopic aspect and microscopic examination.
Chi-square analyses were used to analyze the different distribution of tinea capitis among boys and girls, different age groups and the different schools.

Results

The figure shows examples of clinical fungal infection on the scalp in the study population. Table 1 provides the baseline characteristics of the 454 schoolchildren who participated in the study. Of the 454 children examined during the survey, a total of 105 (23.1 %) had clinical signs of tinea capitis (Table 1). Direct examination (KOH) was performed in 97 of these 105 children and 1 time in a child without clinical suspicion of

Table 1 Demographic data for tinea capitis among schoolchildren in Gabon.

	N	Positive by physical examination N (%)	Positive by KOH N (%)	Positive by Culture N (%)	Positive by KOH and/or culture N (%)
All children together	454	105 (23.1)	60 (13.2)*	69 (15.2)**	74 (16.3)
Gender					
Male	227	58 (25.6)	35 (15.4)	38 (16.7)	41 (18.1)
Female	227	47 (20.7)	25 (11.0)	31 (13.7)	33 (14.5)
Age (years)					
4-9	262	71 (27.1)#1	43 (16.4)#2	48 (18.3)#3	51 (19.5)#4
10-12	123	24 (19.5)	11 (8.9)	14 (11.4)	15 (12.2)
13-17	69	10 (14.5)	6 (8.7)	7 (10.1)	8 (11.6)
Schools					
Lalala (urban, higher SES***)	245	50 (20.4)	29 (11.8)	30 (12.2)	33 (13.5)
Zile (rural, low SES***)	209	55 (26.3)	31 (14.8)	39 (18.7)	41 (16.9)

SES,socio-economic status.
*There was a clinical suspicion of tinea capitis eight times, but KOH was not performed.
**There was a clinical suspicion of tinea capitis three times, but culture was not performed.
#1p = 0.047, #2p = 0.078, #3 p = 0.102, #4 p = 0.073.

tinea capitis. Culture was performed in 102 children. In total 74 (16.3%) patients were positive for both KOH and / or culture, 60 (13.2%) were positive by KOH and 69 (15.2%) by culture (Table 1).

Tinea capitis was slightly, but statistically non significant, more prevalent among boys compared to girls and in the age group of 4 to 9 years compared to the 13 to 17 years age group (Table 1). The prevalence of tinea capitis did not differ much in the rural school compared to the urban school with a higher socioeconomic status.

The frequency distribution of the different species among the different schools is depicted in Table 2. Combining the data of both schools, the most frequently detected species were *T. soudanense*, *T. tonsurans* and *M. audouinii*. The frequency distribution of the different species was different between the two schools (Table 2). Infections with *T. tonsurans* and *M. audouinii* were more prominent in the rural school while *T. soudanense* was more frequently seen in the urban school.

Table 2 Results of the 102 cultures according to the different schools.

	Schools		Distribution	
Isolated species*	**Urban (Lalala)**	**Rural (Zile)**	**Total**	**(%)****
Total number of cultures	50	52	102	
Isolation of fungal species	30	38	68	100.0
T. soudanense	14	6	20	29.4
T. tonsurans	6	13	19	27.9
M. audouinii	4	13	17	25.0
T. violaceum	2	4	6	8.8
T. not determined	1	1	2	2.9
T. rubrum	1	0	1	1.5
M. canis	0	1	1	1.5
T. equinum	1	0	1	1.5
T. mentagrophytes	1	0	1	1.5
Results of other cultures				
Saprophyte	2	2	4	
Bacteria	0	0	0	
Sterile	18	11	29	

*T = Trichophyton; M = Microsporum.
**Frequency distribution among the 68 cultures with isolation of fungal species.

Table 3 summarizes the previous prevalence studies on fungal cultures in schoolchildren carried out in different sub-Saharan African countries and compares those with the present study.[1,7-15]

Discussion

Our study showed that clinically manifest tinea capitis is endemic among schoolchildren in the Lambaréné region, Gabon. The overall prevalence of clinically manifest tinea capitis was 23% and varied from 20% in the urban school with a high socioeconomic status to 26% in the rural school with lower socioeconomic status. The slightly higher prevalence of tinea capitis in the rural school might be explained by a lower availability of antifungal treatments, poor hygienic conditions, or school and household over-crowding. A same pattern was observed in our study in the Greater Accra region in Ghana.[10]

In our studies we did not test for minimal infection, termed carrier state, i.e. we did not collect samples from all children. We, therefore, may have missed some children with asymptomatic dermatophyte scalp carriage so that the real prevalence of tinea capitis may even be higher. Anthropophilic dermatophytes (i.e. *T. tonsurans* and *T. violaceum*) have been generally associated with high rates of asymptomatic carriage.[16]

Like in other studies performed in the region *T. soudanense* appeared to be one of the most frequent causative agents.[2,11,17-19] The percentage of *T. violaceum* (8.8%) was much lower than recent figures found in South Africa (90%),[20] Ghana (26%),[10] Ethiopia and Rwanda (42%),[21,22] although in a study in Mozambique also very few cases of *T. violaceum* were found.[15] These prevalences are different compared to those provided in an excellent review dating from 1974, in which *T. violaceum* was more prevalent in Northern and Eastern Africa (25% or more frequent).[19] Changes in geographical distribution over time may be related to increasing mobility of the population. The percentage of *M. audouinii* (25%) was comparable to other studies performed in Africa.[1,9,11,13,15,22] In other studies from Nigeria and Ghana *M. ferrugineum* was present in a high percentage (between 7.7-17.3%),[1,7,10] while in the present study this species was not found. The percentages of infections with both *T. tonsurans* and *M. audouinii* were high (27.9% and 25.0%). There does not appear to be a replacement of *M. audouinii* by *T. tonsurans* as has been seen in the past half century in the United States which might have been caused by the success of griseofulvin treatment on the US mainland.[20]

The prevalence of tinea capitis was highest among boys in the younger age group (4-9 years) as reported in earlier studies,[1,2,11,17] although in our study statistical significance was not reached. The spectrum of fungal species varied considerably between the schools. In the rural school with a lower socioeconomic status the percentage of *M. audouinii* was higher and the percentage of *T. soudanense* lower than the urban school

Table 3 Summary of cross sectional studies on tinea capitis in school children carried out in sub-Saharan African countries

Study description	Eastern / SouthernAfrica			Western Africa							
First author	Ayaya	Robertson	Sidat	Menan	Oyeka	Enweani	Nweze	Emele	Ayanbimpe	Hogewoning	Current study
Country[reference]	Kenya[8]	Zimbabwe[14]	Mozambique[15]	Ivory Coast[11]	Nigeria[13]	Nigeria[9]	Nigeria[12]	Nigeria[1]	Nigeria[7]	Ghana[10]	Gabon
Year(s) of study	2001	1990	2001	1998-99	1984	1996	1997-98	2002-2005	2004	2004	2005
Number of children	68	704	685	1913	1555	1400	2193	47723	28505	463	454
Age (years)	6 to 14	5 to 9	4 to 15	4 to 15	4 to 18		4 to 16	2 to 15	3 to 16	5 to 17	4 to 17
Prevalence tinea capitis											
Clinical positive	33.3%	202 (29.0%)	67 (9.8%)	227 (11.8%)	300 (19.3%)			4498 (9.4%)	796 (2.8%)	39 (8.4%)	105 (23.1%)
KOH positive		149 (21.2%)		211 (11.0%)						27 (5.8%)	60 (13.2%)
Culture positive		140 (19.9%)	67 (9.8%)	217 (11.3%)	158 (10.2%)	188 (13.4%)		502 (1.1%)		27 (5.8%)	69 (15.2%)
KOH and/or culture positive		174 (24.5%)	67 (9.8%)	220 (11.5%)			154 (7.0%)	502 (1.1%)	248 (0.9%)	31 (6.7%)	74 (16.3%)
Mycological results											
Trichophyton											
T. *concentricum*										7.4%	
T. *ferrugineum*								17.3%	7.7%	7.4%	
T. *interdigitale*		3.8%									
T. *mentagrophytes*		1.4%	29.9%		24.0%	25.5%	16.7%	15.7%	7.3%		1.5%
T. *rubrum*[1]	4.0%					21.3%		0.2%	5.2%	11.1%	1.5%
T. *schoenlenii*					9.0%		28.1%	3.8%	1.6%		
T. *soudanense*[1]				63.6%	15.0%			8.0%	30.6%		29,40%
T. *tonsurans*	77.8%				9.0%	2.1%	10.5%	0.8%	7.3%	22.2%	27.9%
T. *verrucosum*							20.2%	1.0%			
T. *violaceum*		78.0%		2.3%				0.8%	6.5%	25.9%	8.8%
T.*equinum*											1.5%
T. *yaoundei*		0.1%					4.4%	2.6%			
Microsporum											
M. *audouinii*[2]		7.1%	86.6%		38.0%	46.8%		42.2%	7.7%	14.9%	24,60%
M. *canis*		2.1%							7.3%		1.5%
M. *gallinae*							18.4%				
M. *gypseum*			3.0%		5.0%		1.8%		6.9%	3.7%	
M. *langeronii*[2]				31.3%							
Other											
Epidermophyton floccosum						4.3%					
F. solani										7.4%	
Mixed agents		7.9%		2.8%				5.8%			

T. *Trichophyton* ; M. *Microsporon* ; F. *Fusarium* .

[1]T.*rubrum* and T. *soudanense* are now considered the same species.

[2]M.*audouinii* and M.*langeronii* are also considered the same species. Some percentages add up to more than 100% because of double infections.

with a higher socioeconomic status, a pattern seen before in Ghana and Nigeria.[7,10] It should be noted, however, that prevalence data of studies performed in one area of the country cannot be generalized to the entire country.[2,7]

Different clinical presentations of tinea capitis were sparse, most children presented with a non-inflammatory type of tinea capitis especially 'gray-patch' scaling alopecia, seborrheic-dermatitis like scales and patches of 'black dot' and 'chicken skin' alopecia. We only saw one child with a kerion, an inflammatory form of tinea capitis, which can result in alopecia with scarring.

In Conclusion, our study showed that tinea capitis is endemic among schoolchildren in the Lambaréné region in Gabon and that the prevalence to a certain extent depended on the location and the socio-economic status of the school that was investigated. In addition, there were important varieties in the causative species described between the different schools in our study group and the causative species were different compared to studies conducted in other African countries.

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Chapter 5

Point and period prevalences of eczema in rural and urban schoolchildren in Ghana, Gabon and Rwanda

Journal of the European Academy of Dermatology and Venereology
Volume: 26, Issue: 4 Date: 2012 Apr, Pages: 488-94

AA Hogewoning [1,2,3], JN Bouwes Bavinck [3], AS Amoah [5], DA Boakye [5], M Yazdanbakhsh [4], PG Kremsner [7,8], AA Adegnika [4,7,8], SKAD De Smedt [6], R.Willemze [3], APM Lavrijsen [3]

[1] Dermatology, University of Ghana Medical School, Korle-Bu Teaching Hospital, Accra, Ghana
[2] Dermatology, King Faisal Hospital, Kigali, Rwanda
[3] Dermatology, Leiden University Medical Center, Leiden, The Netherlands
[4] Parasitology, Leiden University Medical Center, Leiden, The Netherlands
[5] Noguchi Memorial Institute for Medical research, University of Ghana, Legon, Ghana
[6] Ophthalmology, Kabgayi Hospital Rwanda
[7] Medical Research Unit, Albert Schweitzer Hospital, Lambaréné, Gabon
[8] Institute for Tropical Medicine, University of Tübingen, Tübingen, Germany

Abstract

Background
Eczema is a growing problem in Africa, particularly amongst children.

Objective
To investigate the point-prevalences of eczema by physical examination in school-children living in rural and urban areas and with different socioeconomic backgrounds in Ghana, Gabon and Rwanda. In Ghana period-prevalences were also astimated by questionnaire and compared with the point-prevalences.

Methods
In total, 4839 schoolchildren in Ghana, Gabon and Rwanda were seen by at least one dermatologist. The point-prevalences of eczema were estimated on the basis of physical examination. Period-prevalences were measured in Ghana with questionnaire based-interviews adapted from the International Study of Asthma and Allergies in Childhood (ISAAC).

Results
The point-prevalences were 1.5% and 1.6% in the two Ghanaian studies; 4% in Gabon and 0.8% in Rwanda. The period-prevalences were 2.6% and 4.4% in the two Ghanaian studies. The prevalences of eczema were not significantly different when comparing the urban and rural groups as well as the different socio economic levels. The sensitivity and positive predictive value to identify eczema cases based on the questionnaires compared to the diagnoses by physical examination were only 33% and 22% in the first Ghanaian study and 10% and 4% in the second Ghanaian study, respectively.

Conclusions
The point-prevalences of eczema in the three African countries studied were low compared with industrialized countries. Physical examination by a dermatologist still is the gold standard to identify eczema cases because the sensitivity and the positive predictive value to identify eczema cases with questionnaires were low in the two Ghanaian studies.

Conflict of interest
The authors state no conflict of interest.

Introduction

Eczema, also called atopic eczema, flexural eczema, atopic dermatitis, neurodermitis or atopiform dermatitis, is a chronic relapsing inflammatory skin disorder which is becoming increasingly common worldwide.[1-3] Because nearly 60 percent of patients do have clinical features without IgE-mediated sensitivity to allergens the term eczema instead of atopic eczema is preferred.[4-7]

Physical examination by a dermatologist or a team of dermatologists is still the gold standard to diagnose eczema.[4;5] The diagnosis of eczema is based on a constellation of clinical findings. The most frequently used clinical criteria for the diagnosis of eczema are those of Hanifin and Rajka.[8] These criteria are based on clinical experience and were published in 1980 after a consensus conference. They have since then been widely used in genetic, biological, immunological and epidemiological studies. The U.K. Working Party has tried to refine the criteria of Hanifin and Rajka by developing a minimal set of five diagnostic criteria especially suitable for epidemiological purposes.[9] In addition the Millennium Criteria were developed in which the presence of allergen-specific IgE in a patient is required for making the diagnosis of atopic eczema. In the Millennium Criteria the term atopiform dermatitis is reserved for the patients with the clinical phenotype but without detectable allergen-specific IgE.[2;7;9]

Questionnaires are often used in epidemiological studies to identify persons with eczema. The main advantages of questionnaire-based prevalence studies are that questionnaires are relatively cheap to distribute and administer to children and their parents and / or caretakers. In addition, questionnaires can be used to measure disease prevalences in large populations. A major shortcoming of questionnaires however, is the fact that eczema is difficult to define because of its variable morphology, distribution and its intermittent nature.[2;3;10;11] Many itchy, scaly and erythematous skin diseases are judged by non dermatologists as being eczema which may lead to an overestimation of eczema.

Physical examination measures a point-prevalence while a questionnaire can also measure a period-prevalence. Because eczema is a chronic relapsing disorder a period-prevalence may be the best method to determine its frequency. When one measures a point-prevalence there is always a chance of underestimation.[5-7;12]

The main purpose of our study was to estimate the point -prevalences of eczema as diagnosed by a dermatologist in schoolchildren living in rural and urban areas and with different socioeconomic backgrounds in Ghana, Gabon and Rwanda. In Ghana we also estimated the period prevalences by questionnaire and compared these with the point prevalences by physical examination. A selection of articles reporting prevalence data amongst sub-Saharan African schoolchildren is supplied to provide a framework for the prevalence data of our study.

Materials and Methods

To provide information about the prevalence of eczema in West and Central Africa, cross sectional studies with 4839 schoolchildren were conducted between 2004 and 2007 in Ghana (2 studies), Gabon and Rwanda. Details of the studies are presented in Table 1.

Point-prevalences of eczema were determined in all four studies by physical examination of all children by at least one dermatologist or a team of dermatologists (Table 1). Period-prevalences were measured by questionnaires adapted from the International Study of Asthma and Allergies in Childhood (ISAAC) in Ghana by trained staff with one of the caretakers of each child. The three most important questions are detailed in Figure 1. Figures 2-4 show clinical pictures of the children with eczema.

Independent persons with advanced levels of English language and mother tongue of Twi, Dangme, Ga or Ewe translated the questionnaire into these languages. In Ghana the rural and urban areas were selected according to the guidelines of the Ghana Statistical Service. This service defines an urban area as a settlement with a population greater than 5000 individuals. Our target urban area was the Accra Metropolitan Area with an estimated population of 1 801 606 in 2002. Our rural target areas were all settlements some distance from the Accra Metropolitan Area with low population densities. Other defining criteria for our rural areas included; settlements where the main income generating activities revolved around agriculture (fishing or farming), areas with limited access to amenities such as water, electricity, health, education, transportation and communication.

In Rwanda and Gabon rural schools were situated in villages, remote from the main road with lower population density than the urban areas. The urban schools were located in the middle of the capital city or one of the major towns of the country, close to the main roads.

Schools were selected for recruitment based on a broad social economic classification. This broad social economic classification reflects the average socioeconomic level (SEL), respectively low, middle and high. Urban high SEL schools were private fee-paying schools while urban low schools were public, government-run schools where no school fees were paid.

Categorical data were analyzed for statistical differences by chi-square test. Point prevalences and period prevalences were estimated by calculating the proportions of children with eczema compared to the total groups of children with 95% confidence intervals. Sensitivity, specificity and predictive values were calculated according to standard methods. For the statistical analyses, we used SPSS for Windows version 16.0 (SPSS Inc, Chicago, IL, USA) and software which is freely available on the internet (http://www.dimensionresearch.com/resources/calculators/conf_prop.html).

Table 1 Characteristics of the studies.

	GHANA (1)	GHANA (2)	GABON	RWANDA
Region	Greater Accra Region: Accra Metropolitan Area and Ga West District	Greater Accra Region: Accra Metropolitan Area , Dangme East District and Ga East District	Albert Schweitzer Hospital which is located about 6 km from the city center of Lambaréné	Muhanga (Gitarama and Saki), Bugesera (Gicaca) and Kicuciro (Gicondo, Kigali)
Number of schools				
- Rural public (low SEL*)	2	6	1	3
- Urban public (low SEL)	1	3	1	3
- Urban private (middle SEL)	0	1	0	0
- Urban private (high SEL)	1	1	0	0
Part of these studies	Association of helminth infection with allergic sensitization and atopic eczema among schoolchildren. In cooperation with the department of Parasitology, Leiden University Medical Center.	EU project GLOFAL "Global view of food allergy: opportunities to study the influence of microbial exposure". In cooperation with the department of Parasitology, Leiden University Medical Center.	Association of helminth infection with allergic sensitization and atopic eczema among schoolchildren. In cooperation with the department of Parasitology, Leiden University Medical Center.	Prevalence of Vernal Keratoconjunctivitis in Rwandan schoolchildren and its association with atopy and parasitic infestation. In cooperation with the department of ophthalmology, Medical University Gent.

*SEL: socioeconomic level.

Figure 1

Three most important questions for a history of symptoms of eczema (period-prevalence) translated from English in Twi, Dangme, Ga, and Ewe (Ghana).

(Q1) Has your child ever had an itchy rash which was coming and going for at least six months?

If Q1 was answered with yes:

(Q2) Has your child had this itchy rash at any time in the past 12 months?

(Q3) Has this itchy rash at any time affected any of the following places: the folds of the elbows, behind the knees, in front of the ankles, under the buttocks, or around the neck, ears or eyes?

The child was considered to have a history of symptoms of eczema if the last two questions were answered confirmative ([12]).

Figure 2 Danny Morgan folds, hyper-pigmentation in the face and anterior neck folds.

An overview of the recent literature is provided after searching Pubmed for the words 'atopic dermatitis' and 'Africa'. The relevant articles were selected and the references in these articles were checked for additional studies. The prevalence data for schoolchildren were extracted and summarized. Non-English articles and hospital-based prevalence data were excluded. The aim of this literature search was not to perform a systematic review of the prevalence of eczema in sub-Saharan Africa but to provide a framework of other recent studies in which we could place and compare the prevalence data of our own study.

Figure 3 Knee folds with typical localization of hyperpigmentation and lichenification.

Figure 4 Prurigo and scratch marks with dry skin and hyperpigmentation.

Results

The baseline characteristics of the children are presented in Table 2. Most children were between 4 and 16 years old and boys and girls were equally distributed (Table 2).

Table 2 Baseline characteristics of the children in the different countries.

	GHANA (1) N (%)	GHANA (2) N (%)	GABON N (%)	RWANDA N (%)
Number of children	463	1394	454	2528
Age distribution				
4-8	128 (27.6)	299 (21.4)	197 (43.4)	327 (12.9)*
9-12	275 (59.4)	804 (57.7)	188 (41.4)	1494 (59.1)
13-16	36 (7.8)	282 (20.2)	68 (15.0)	707 (28.0)
17-20	0	9 (0.6)	1 (0.2)	0
unknown	24 (5.2)	0	0	0
Sex				
Girls	201 (43.3)	734 (52.7)	227 (50.0)	1296 (51.3)
Boys	262 (56.6)	660 (47.3)	227 (50.0)	1224 (48.4)
Unknown	0	0	0	8 (0.3)
Characteristics of the schools				
Rural public (low SEL**)	226 (48.8)	753 (54.0)	209 (46.0)	1455 (57.6)
Urban public (low SEL)	125 (27.0)	214 (15.4)	245 (54.0)	1073 (42.4)
Urban private (middle SEL)	0	356 (25.5)	0	0
Urban private (high SEL)	112 (24.2)	71 (5.1)	0	0

*In Rwanda the youngest child was 8 years old.
** SEL: socioeconomic level.

The point-prevalences of eczema based on physical examination by a dermatologist are presented in Table 3 and range between 0.8% in Rwanda to 4.0% in Gabon. The point-prevalences were slightly higher in the urban schools compared to the rural schools with a prevalence reaching 4.5% in the urban private school with high socioeconomic level in the first Ghanaian study (5 out of 7 children with eczema were found in the urban private rich school), but statistical significance was not reached (Table 3). In contrast, in Gabon the point-prevalence of eczema was higher in the rural school (5.7 %) compared to the urban school (2.5 %) (Table3).
Questionnaires were available from 345 (74.9%) children of the first Ghanaian study and 1245 (89.3%) of the second Ghanaian study. The period-prevalences based on these questionnaires are also presented in Table 3. The sensitivity, specificity and the positive and negative predictive values of the diagnoses of eczema based on questionnaires in

Table 3 Point-prevalence of atopic dermatitis based on physical examination by a dermatologist and period-prevalence based on the questionnaires in the different countries.

	GHANA (1) n/N % (95% CI)	GHANA (2) n/N % (95% CI)	GABON n/N % (95% CI)	RWANDA n/N % (95% CI)
Point prevalence based on physical examination	7/463 1.5 (0.4;2.6)	22/1394 1.6 (0.9;2.2)	18/454 4.0 (2.2;5.8)	20/2528 0.8 (0.4;1.1)
Point prevalence stratified according to the schools				
Rural (low SEL*)	2/226 0.9 (0;2.1)	10/753 1.3 (0.5;2.2)	12/209 5.7 (2.6;8.0)	10/1455 0.7 (0.3;1.1)
Urban combined	5/237 2.1 (0.3;3.9)	12/641 1.9 (0.8;2.9)	6/245 2.5 (0.5;4.4)	10/1073 0.9 (0.4;1.5)
Urban (low SEL)	*0/125 0 (0;2.3)*	*2/214 0.9 (0;2.2)*	*6/245 2.5 (0.5;4.4)*	*10/1073 0.9 (0.4;1.5)*
Urban private (middle SEL)	--	*8/356 2.3 (0.7;3.8)*	--	--
Urban private (high SEL)	*5/112 4.5 (0.6;8.3)*	*2/71 2.8 (0;6.7)*	--	--
Period prevalence based on questionnaire (flexural eczema)	9/345 2.6 (0.9;4.3)	55/1245 4.4 (3.3;5.6)	Not measured	Not measured

*SEL: socioeconomic level; CI: confidence interval.

relation to the diagnoses based on physical examination are presented in Table 4. Table 5 shows examples of population-based point- and period-prevalences in sub-Saharan Africa of recent studies in comparison with the current study.

Table 4 Sensitivity and specificity of the diagnoses of eczema based on questionnaires in relation to the diagnoses based on physical examination (reference standard).

	GHANA (1)		GHANA (2)	
	Eczema by physical examination	No eczema by physical examination	Eczema by physical examination	No eczema by physical examination
Eczema by questionnaire	2	7	2	53
No eczema by questionnaire	4	332	18	1172
Total	6	339	20	1225
Sensitivity	2/6 33 (0; 71.1)		2/20 10 (0;23.2)	
Specificity	332/339 98 (96.4; 99.5)		1172/1225 96 (94.5; 96.8)	
Positive predictive value	2/9 22 (0; 49.4)		2/55 4 (0; 8.6)	
Negative predictive value	332/336 99 (97.7 ; 100)		1172/1190 98 (97.8 ; 99.2)	

Table 5 Population-based point- and period-prevalences among schoolchildren in sub-Saharan Africa.

			Point-prevalence by physical examination		Period-prevalence by questionnaire	
Country	Year	References	n/N	% (95% CI)	n/N	% (95% CI)
Cameroon	2007	ISAAC[16]			15/215	7.2% (3.7;10.7)
Congo	2007	ISAAC[16]			26/164	16.2% (10.421.6)
	2007	ISAAC[16]			35/320	10.9% (7.6;14.4)
Guinea	2007	ISAAC[16]			112/587	18.8% (15.8;22.2)
Ivory Coast	2007	ISAAC[16]			109/607	18.2% (14.9;21.1)
Ethiopia	2005	Haileamlak[11]	79/7915	1.8% (1.5;2.1)	348/7915	4.4% (4.0;4.9)
	2007	ISAAC[16]			115/606	19.0% (15.9;22.1)
Ghana	2009	ISAAC[12]	5/1325	0.4% (0;0.7)	40/1325	3.5% (2.1;3.9)

Table 5 Continued.

			Point-prevalence by physical examination		Period-prevalence by questionnaire	
Country	Year	References	n/N	% (95% CI)	n/N	% (95% CI)
Ghana	2004	This study	7/463	1.5% (0.4;2.6)	9/345	2.6% (0.9;4.3)
	2007	This study	22/1394	1.6% (0.9;2.2)	55/1245	4.4% (3.3;5.6)
Gabon	2007	ISAAC[16]			65/454	14.4% (11.1;17.5)
	2005	This study	18/454	4.0% (2.2;5.8)		
Kenya	2007	ISAAC[16]			81/509	15.5% (12.8;19.2)
	2007	ISAAC[16]			66/449	14.9% (11.4;18.0)
Mozambique	2007	Mavale-Manuel[22]			466/5013	9.3% (8.5;10.1)
Nigeria	2005	Ogunbiyi[26]	0/1066	0% (0;0.3)		
	2007	ISAAC[16]			19/241	7.9% (4.3;11.1)
Rwanda	2007	This study	20/2528	0.8% (0.4;1.1)		
South Africa	1995	Zar[18]			611/5178	11.8% (10.9;12.7)
	2002	Zar[18]			9775037	19.4% (18.3;20.5)
	2007	Chalmer[31]	31/3067	1.0% (0.6;1.4)	55/ 3067	1.8% (1.3 ;2.2)
	2007	ISAAC[16]			58/520	11.2% (8.4;13.9)
	2007	ISAAC[16]			89/670	13.3% (10.7;15.9)
	2007	ISAAC[16]			401/2996	13.4% (12.2;14.6)
Sudan	2007	ISAAC[16]			6/137	4.7% (1.2;8.2)
Togo	2007	ISAAC[16]			36/332	10.7% (7.5;14.2)

Discussion

The prevalence of eczema in the industrialized world has increased rapidly in the last few decades. [2,13-15] Surveys conducted worldwide revealed period-prevalences between 15% and 30% in different age groups of children. Most of these data were obtained by questionnaire.[2,13] Generally, high prevalences were found in the developed countries such as Northern Europe, North America, Japan and Australasia and low prevalences in countries such as China, Iran and Ethiopia.[2,6,11,13,16,17] Although the prevalence rates of eczema in the developing world are much lower, recent studies show a sharp increase due to rapid urbanization, changes in lifestyle and socioeconomic status as well as frequent washing.[2,13,18–20] In West Africa, the prevalence of eczema has been considered

to be less than 5%,[21–23] though recent studies in this region as well as other parts of Africa have shown an increase,particularly amongst infants.[24–26] Most of the latter studies, however, were hospital-based and therefore are less reliable when estimating the prevalence of eczema on a national scale.[21,23,25,27–29]

The point-prevalences in our study of eczema amongst schoolchildren as measured by physical examination by a dermatologist in Ghana (1.5% and 1.6%), Gabon (4.0%) and Rwanda (0.8%) were comparable with other studies in sub-Saharan Africa (Table 5). It was notable that the point prevalence of eczema was significantly higher in the rural area of Gabon, for which we do not have a clear explanation. The point-prevalences in our study did not differ significantly between rural and urban areas, which is in agreement with a recent overview about differences between point-and period-prevalences of eczema in rural and urban areas in developing countries.[13] This may be explained by the fact that rural and urban areas in sub-Saharan Africa are much more comparable than in Western countries.

The questionnaire based period-prevalences in Ghana were about twofold higher than the point-prevalences as measured by physical examination, which can be explained by the chronic relapsing character of eczema.[5–7,12] The sensitivity and the positive predictive values of the questionnaires, however, were low (33% and 22% in the first and 10% and 4% in the second Ghanaian study respectively).

The questionnaire-based period-prevalences in other sub-Saharan African countries were generally more than 10% (Table 5). Flohr et al. recently showed a poor correlation between the ISAAC questionnaires and the presence of permanent flexural eczema in non-English speaking and developing countries, although there was a good correlation between these two measurements amongst Anglophone children.[12] Most probably these discrepancies are caused by factors related to translation of the questionnaires from the English language as well as cultural and educational differences in developing countries.[12,30–32] It could be queried whether the questionnaire-based period-prevalences in the other sub-Saharan African countries are real or may be an overestimation due to difficulties in the definition of eczema.

The strength of our study is that we included thousands of schoolchildren in three different countries, that all children were seen by a dermatologist or a team of dermatologists and that the definition of rural and urban schools within the countries was the same. A potential limitation of our study is that we only evaluated the period prevalence by questionnaire in Ghana.

Our data indicate that in African countries the use of questionnaires one such as the ISAAC is a less reliable method to identify children with eczema.[11,12,31] Probably physical examination is a better method to measure the prevalence of eczema amongst children in these countries. It must be realized, however, that physical examination only measures the point-prevalence and may give an underestimation of the real prevalence of a disease that 'comes and goes' over time, such as eczema. Therefore, the gold standard

to identify children with eczema remains, physical examination by a dermatologist or team of dermatologists, especially when the aim of the study is to find cases to analyse risk factors for eczema or to do genetic research.[10–12]

Acknowledgements

The authors thank I. Larbi, Y. Aryeetey, B. Obeng and Y. Fonteyne for their support. The study was made possible by a gift from the Gratama Foundation, the Netherlands, by EU-project GLOFAL 'Global view of food allergy: opportunities to study the influence of microbial exposure' FP6-2003-Food-2B contract: FOOD-CT-2005-517812, by The Netherland Organization for Scientific Research for Global Development, WOTRO grant number WB 93-433 and by the cooperation with the University of Gent, Belgium. We thank all children, parents, teachers and fieldworkers for their participation.

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Chapter 6

Allergic characteristics of urban schoolchildren with atopic eczema in Ghana

Journal of the European Academy of Dermatology and Venereology
Volume: 24, Issue: 12, Date: 2010 Dec, Pages: 1406-12

Hogewoning A.A.,[1,5] Larbi I.A.,[2] Addo H.A.,[1] Amoah A.S.,[2]
Boakye D.,[2] Hartgers F.,[3] Yazdanbakhsh M.,[3] van Ree R.,[4] Bouwes Bavinck J.N.,[5]
Lavrijsen A.P.M.[5]

[1] Dermatology, University of Ghana Medical School, Korle-Bu Teaching Hospital, Accra, Ghana,
[2] Noguchi Memorial Institute for Medical Research, University of Ghana, Legon, Ghana,
[3] Parasitology, Leiden University Medical Centre, Leiden, The Netherlands,
[4] Department of Experimental Immunology and Department of Otorhinolaryngology,
Academic Medical Centre - University of Amsterdam, Amsterdam, The Netherlands,
[5] Dermatology, Leiden University Medical Centre, Leiden, The Netherlands

Abstract

Background
Atopic eczema is an increasing clinical problem in Africa.

Objective
To determine allergic characteristics and to identify possible risk factors for eczema among schoolchildren in an urbanized area in Ghana.

Patients and Methods
Schoolchildren aged 3-16 years with eczema were recruited. For each patient, one to three age and sex-matched controls were selected. All children completed a questionnaire and were skin prick tested with a panel of allergens. Blood was drawn to determine total and allergen-specific IgE. Conditional logistic regression models with the matching factors included in the model wre used to calculate the odds ratios and to adjust for possible confounders.

Results
A total of 52 children with eczema (27 boys and 25 girls) and 99 controls were included. Levels of total IgE were found to be 9.1 (1.1; 78.4) times more often elevated in children with eczema. This association was mainly driven by elevated IgE levels against cockroach antigen. Children with eczema were found to have 2.0 (0.87; 4.7) times more often positive skin prick tests (SPT), but this association diminished to 1.2 (0.40; 3.6) after adjustment for total IgE levels. Frequent washing with soap was identified as a risk factor for the development of eczema among these children.

Conclusion
Schoolchildren with eczema in Ghana were characterised by elevated IgE levels especially against cockroach antigen. The association between eczema and positive SPT was much weaker suggesting immune hyporesponsiveness of the skin. After adjustment for IgE level, SPT were less suitable to distinguish children with and without eczema.

Conflict of interest
The authors state no conflict of interest.

Funding
The study was made possible by a gift from the Gratama Foundation, the Netherlands.

Introduction

Atopic dermatitis is a chronic relapsing pruritic inflammatory skin disorder.[1-3] Although termed atopic, up to 60 percent of children with the clinical phenotype do not have demonstrable IgE-mediated sensitivity to allergens.[2] Therefore, we prefer to use the term "eczema" throughout this paper instead of "atopic dermatitis." We have used the criteria of Hanifin and Rajka[4,5] to define our cases with "eczema". Eczema is widespread and its prevalence is rapidly increasing in the industrialized world.[2,6] Eczema is also a growing clinical problem in developing countries. In West Africa the prevalence of eczema was considered to be < 5%,[7-11] though recent studies in West Africa and other parts of Africa have shown an increase, particularly amongst infants,[12-15] perhaps because of the improvement of diagnostic measures.

The rising prevalence of eczema might be related to improved sanitation and reduction in childhood infections in developed countries, which is known as the hygiene hypothesis.[16-19] According to this hypothesis, bacterial and viral infections in early life result in the proper maturation of the immune system, thereby reducing the expression of pro-allergic T-helper-2 (Th2) responses.[19-21] Parasitic infections have also been shown to induce hyporesponsiveness and to be negatively associated with clinical atopy or allergy,[22,23] possibly due to suppression of mast cell function.[24]

Risk factors for eczema which are related to the hygiene hypothesis are increasing gross national per-capita income,[11] changes in lifestyle due to a higher socio-economic status,[25] reduced crowding at home,[26] sleeping on mattresses, changes in food consumption patterns, and more frequent washing.[6,19,27] Eradication of endoparasites, vaccinations and the treatment of other infections may also increase the risk of eczema.[22,23] Frequent washing can affect the skin barrier function, providing a non-immunological explanation why frequent washing may increase the risk of eczema.[19,28-30]

The growing urbanization in Africa has been associated with an increased risk of eczema,[31] supporting the fact that changes in the environment and habits may be resulting in the allergic march, as seen in highly developed countries.

The role of atopy in the pathophysiology of eczema is still under debate.[1,32] One hypothesis states that immunological disturbance causes IgE-mediated sensitization resulting in epithelial-barrier dysfunction as a consequence of local inflammation; the second hypothesis proposes an intrinsic defect in the epithelial cells, leading to barrier dysfunction.[19,28-30] In the latter model, the immunologic aspects are secondary to the epithelial-barrier dysfunction and could be considered as an epiphenomenon.[19,28-30]

The purpose of this matched case-control study was to determine the allergic characteristics of urban African schoolchildren and to identify possible risk factors for eczema in these children.

Patients and Methods

Study design

Between February and December 2005, 86 schoolchildren with moderate to severe eczema were selected at the dermatological out-patient clinics of 3 hospitals in Accra, Ghana by a dermatologist (A.H.) according to the criteria as defined by Hanifin and Rajka.[4,5] For each included child with eczema two controls matched for age and sex without any visible symptoms of eczema were selected from the same school and class.

Study Area

Both the hospitals and schools are located in the urbanized area of the Greater Accra region of Ghana, West Africa. The study area's climate is warm with the rainy season in April to June and September to November.

Informed Consent and Ethical approval

Information sheets and consent forms were given to the cases and potential controls in school to take home to their parents. Interested parents filled the consent forms and returned them to the schools. Ethical approval was granted by the Institutional Review Board of the Noguchi Memorial Institute for Medical Research. The ethical approval number was CPN015/02-03.

Questionnaires

Questionnaire-based interviews were completed by trained staff with one of the care-takers of each child in the study. The questionnaire was adapted from the International Study of Asthma and Allergies in Childhood (ISAAC)[33] and had, among others, questions on symptoms of eczema, asthma and allergic rhino conjunctivitis, and sleeping and washing habits. The most important questions regarding eczema, asthma and allergic rhino conjunctivitis are summarized in Figure 1.

Collection and work-up of samples

All cases and controls were skin prick tested on non-affected skin of the forearm with a panel of allergens (house dust mite (*Dermatophagoides pteronyssinus, D. farinae*), cockroach (*Blatella germenica)*, cat, dog, grass and peanuts) to determine the sensitivity of the study population to specific allergens. Peripheral blood was drawn to collect sera for the determination of the levels of total IgE (enzyme-linked-immunosorbent serologic assay) and allergen-specific IgE Immuno-CAP ™ (Phadia AB, Uppsala, Sweden) in the Netherlands according to the standard protocols.

Figure 1

Three most important questions for a history of symptoms of eczema:

(1) Has your child ever had an itchy rash which was coming and going for at least six months?

(2) Has your child had this itchy rash at any time in the past 12 months?

(3) Has this itchy rash at any time affected any of the following places: the folds of the elbows, behind the knees, in front of the ankles, under the buttocks, or around the neck, ears or eyes?The child was considered to have a history of symptoms of eczemaif all three questions were answered confirmative.

Two most important questions for a history of symptoms of asthma:

(1) Has your child had wheezing or whistling in the chest in the past 12 months?"

(2) In the past 12 months, has wheezing ever been severe enough to limit your child's speech to only one or two words at a time between breaths? The child was considered to have a history of symptoms of asthmaif the first question was answered confirmative and of severe asthma if the second question was also answered confirmative.

Two most important questions for symptoms of allergic rhino conjunctivitis:

(1) In the past 12 months, has your child had a problem with a runny or blocked nose?

(2) In the past 12 months, has this nose problem been accompanied by itchy-watery eyes? The child was considered to have a history of symptoms of allergic rhino conjunctivitis if both questions were answered confirmative.

The presence of malaria parasites was assessed at the Noguchi Memorial Institute, Accra by examining thick and thin blood smears for each participant. Intestinal helminth infection in stool and *Schistosoma haematobium* in urine were assessed at the same centre by standard parasitological examination.

Definition of elevated IgE tests

The values of total IgE were considered elevated when the value was 100 kU/L or higher for children who were 10 years and older; 50 kU/L or higher for children between 7 and 9 years of age; and 25 kU/L or higher for children between 4 and 6 years of age. The values of IgE specific for house dust mite, cockroach, cats dogs, peanuts and grasses were considered elevated when the value was 0.35 kU/L or higher.

Definition of positive skin prick tests

A skin prick test reaction was considered positive when the average of the longest wheal diameter (D1) and its perpendicular length (D2) was >= 3mm for the test allergen

and histamine, while that to the negative control was < 3mm. The results of the skin prick tests were excluded from the analyses when the average histamine response was less than 3 mm and/or the average physiological salt response was 3 or more mm.

Statistical Methods

Logistic regression models were used to calculate odds ratios with 95 percent confidence intervals for the different variables and to adjust for possible confounders as indicated in the Tables. For all statistical analyses we used SPSS for Windows version 16.0 (SPSS Inc, Chicago, IL).

Results

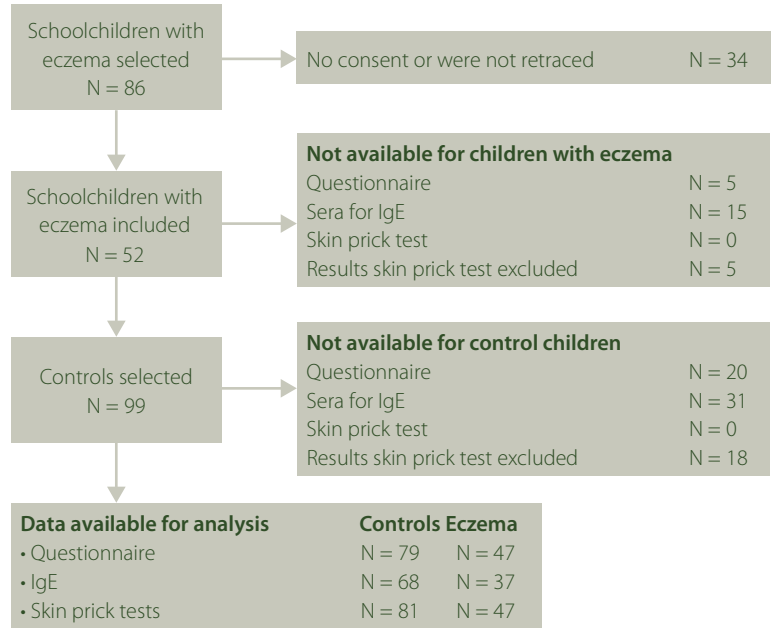
Available and missing data

A total of 52 (60.5%) out of 86 children with eczema who were initially selected for this study could be included (Figure 2). The remaining 34 children who were excluded from the study were those for whom parental consent was not obtained or could not be retraced (they had either left school, changed addresses, or simply could not be found). Accra is a typical big city in a developing country where some people have no fixed addresses and in some areas there is no proper street plan. It was, therefore, often difficult to find the children's residences. In addition, during this study only few of the parents/ caretakers had a mobile telephone, which made retracing the children difficult. Data on missing questionnaires and sera are provided in Figure 2. Skin prick tests were performed in all 52 children with eczema and all 99 control children. The results of five (9.6%) children with eczema and 18 (18.2%) control children were excluded because the response to histamine was less than 3 mm (20 boys and 2 girls) or the physiological salt controls were 3 or more mm in diameter (1 boy). Not all paired eczema-control sets were complete because of missing values for some children with eczema or their paired controls. The numbers of complete data sets of children with eczema and 1, 2 or 3 of their paired controls, respectively, which were used for analysis in the conditional logistic regression model, are provided in Fig. 2.

Baseline characteristics of the children with and without eczema

The baseline characteristics of the children are presented in Table 1. The cases and controls were well-matched. Unintentionally, however, boys with eczema and their controls were significantly younger than the girls with a mean age of the boys with eczema of 8.4 (SD 4.5) compared with a mean age of the girls with eczema of 11.0 (SD 4.2). The difference (with 95% CI) between the age of the boys and girls with eczema was 2.6 (1.3;4.0) years.

Figure 2 Child recruitment flow diagram.



Association of eczema with symptoms of eczema, asthma or allergic rhino conjunctivitis as collected by questionnaire

Using the data from questionnaire most cases reported symptoms of eczema during the last year (P=0.003; Table 2). Although the controls had no visible signs of eczema at the time of investigation still 9.6 % (seven of 73) of the children had reported symptoms of eczema during the last year by questionnaire (Table 2). Symptoms of asthma were not associated with eczema (P=0.555), but symptoms of allergic rhino conjunctivitis were more often reported among cases compared with controls (P=0.051; Table 2).

Association of total and allergen-specific IgE responses with eczema

Children with eczema had significantly more often elevated levels of total IgE than the controls (P=0.023; Table 3). This association was driven by the specific IgE response against cockroach antigen, as in the logistic regression model, the specific IgE responses against house dust mite, peanut and grasses decreased after adjustment for the IgE response against cockroach antigen (Table 3). Performing backward stepwise regression

Table 1 Baseline characteristics of the children with and without eczema.

	Controls (N = 99)	Cases (N = 52)
Sex		
Girls	51 (51.5)	25 (48.1)
Boys	48 (48.5)	27 (51.9)
Age: mean (SD)	10.1 (4.3)	9.7 (4.5)
Age categories (years)		
3 – 5	25 (25.3)	15 (28.8)
6 – 8	16 (16.2)	10 (19.2)
9 – 11	20 (20.2)	9 (17.3)
12 – 14	17 (17.2)	5 (9.6)
15 and older	21 (21.2)	13 (25.0)

Table 2 Association between the clinical characteristics of the children and the answers to the questionnaire.

	Controls (N = 73) n (% Pos)	Cases (N = 41) n (% Pos)	Odds ratios* (95% CI)
Symptoms of eczema during the last year			
No	66 (90.4)	10 (24.4)	1
Yes	7 (9.6)	31 (75.6)	112.9 (4.9 - 2617)
Symptoms of asthma during the last year			
No	62 (84.9)	33 (80.5)	1
Yes	12 (15.2)	8 (19.5)	1.3 (0.5 - ;3.6)
Symptoms of severe asthma during the last year			
No	70 (95.9)	38 (92.7)	1
Yes	3 (4.1)	3 (7.3)	1.9 (0.38 - 9.7)
Symptoms of allergic rhino conjunctivitis during the last year			
No	69 (94.5)	33 (80.5)	1
Yes	4 (5.5)	8 (19.5)	3.4 (1.0 - 11.4)

CI, Confidence Interval
*Odds ratios were calculated with a conditional logistic regression model with the matching factors included in the model.

Table 3 Distribution of elevated IgE levels in children with and without eczema.

	Controls (N = 57) N (% Pos)	Cases (N = 32) N (% Pos)	Odds ratios* (95% CI)	Adjusted odds ratios** (95% CI)
Elevated total IgE #				
No	18 (31.6)	2 (6.2)	1	
Yes	39 (68.4)	30 (93.8)	10.9 (1.4 - 85.0)	
Elevated IgE House dust mite $				
No	45 (78.9)	19 (59.4)	1	1
Yes	12 (21.1)	13 (40.6)	3.2 (1.1 - 9.3)	2.1 (0.53 - 8.2)
Elevated IgE Cockroach $				
No	44 (77.2)	17 (53.1)	1	1
Yes	13 (22.8)	15 (46.9)	3.9 (1.6;9.5)	4.2 (0.92 - 19.1)
Elevated IgE Peanut $				
No	49 (86.0)	26 (81.3)	1	1
Yes	8 (14.0)	6 (18.8)	1.7 (0.54 - 5.3)	1.3 (0.08 - 19.2)
Elevated IgE Grass $				
No	46 (80.7)	24 (75.0)	1	1
Yes	11 (19.3)	8 (25.0)	1.6 (0.56 - 4.3)	0.29 (0.02 - 4.4)

CI Confidence Interval
* Odds ratios were calculated with conditional logistic regression models with the matching factors included in the model.
** Odds ratios are adjusted for the other specific IgE outcomes.
# Elevated total IgE when IgE ≥25 kU/L for 4-6 year old children; ≥50 kU/L for 7-9; ≥100 kU/L for 10 years and older.
$ Specific IgE elevated when IgE ≥ 0.35 kU/L.

only the specific IgE response against cockroach antigen remained in the model resulting in the non-adjusted odds ration as presented in Table 3. Levels of cat- and dog-specific IgE antigens were too low for any conclusions (data not shown).

Association between total IgE responses and skin prick tests

Altogether 28 eczema-control sets (28 children with eczema and 48 controls) had complete data for both IgE and SPT. Not surprisingly, there was a strong association between total IgE responses and SPT (P = 0.009). Of the 57 (75%) children with an elevated total IgE level, 21 (36.8%) showed a positive SPT for any of the antigens tested (38.5% in the children with eczema and 35.5% without eczema: the difference between these 2% was not significant; P = 0.816), whereas only one child in the control group with a non-elevated total IgE level had a positive SPT. Conversely, of the 22 children with or without eczema who had a positive SPT for any of the antigens tested, all except one

Table 4 Distribution of positive skin prick tests in children with and without eczema.

	Controls (N = 81) N (% Pos)	Cases (N = 47) N (% Pos)	Odds ratios* (95% CI)	Adjusted odds ratios# (95% CI)
Any skin prick test (SPT)				
Negative	59 (76.6)	25 (61.0)	1	
Positive	18 (23.4)	16 (39.0)	2.0 (0.87 - 4.7)	
SPT house dust mite				
Negative	66 (85.7)	32 (78.06)	1	1
Positive	11 (14.3)	9 (22.0)	1.5 (0.50 - 4.6)	1.7 (0.52 - 5.2)
SPT cockroach				
Negative	65 (84.4)	36 (87.8)	1	1
Positive	12 (15.6)	5 (12.2)	0.84 (0.29 - 2.8)	0.74 (0.24 - 2.3)

CI, Confidence interval
*Odds ratios were calculated with a conditional logistic regression model with the matching factors included in the model.
Odds ratios are adjusted for the other SPT outcome.

child in the control group had an elevated total IgE level (P = 0.009). Analysing the specific IgE responses and SPT to house dust mite and cockroach antigens gave similar strong associations. The chances of having positive SPT in the presence of elevated IgE levels were 10.5 (1.3; 84.4; P = 0.009) for all tests together and were 82.5 (9.4; 723), P < 0.001 and 10.7 (2.5; 45.9), P < 0.001 for house dust mite and cockroach respectively.

Association between skin prick tests and eczema

A positive SPT for any of the antigens tested was associated with a 2.0 times increased risk of eczema, but statistical significance was not reached (P = 0.104; Table 4). Additional adjustment for total IgE level decreased this association to 1.7 (0.52; 5.2; P = 0.391). There was only a trend of an association with eczema when SPT for house dust mite were considered separately (P = 0.457), and there was no positive association with positive SPT for cockroach antigen (P = 0.755; Table 4). Positivity for SPT with cat, dog, grass and peanuts antigens was too low for any relevant conclusions (data not shown).

Helminth infections and malaria

With the exception of one child with eczema who showed an infection with *Schistosoma haematobium* no infections were detected with *Schistosoma mansoni*, *Ascaris lumbricoides*, *Trichuris*, hookworm, or *Enterobius vermicularis* in either the children with eczema or in the controls.

Malaria was tested in 52 eczema children and 98 controls. Malaria parasites were present in blood smears of 2 (3.8%) cases and 4 (4.1%) controls.

Association of environmental risk factors with eczema

Children with eczema were washing themselves before attending school 4 times more often than the controls and were 2 times more often sleeping on mattresses than their controls without eczema (Table 5). However, both associations did not reach statistical significance (P=0.085 and 0.195; Table 5).

Table 5 Bathing habits and sleeping place of the children in association with eczema.

	Controls (N = 73) N (% Pos)	Cases (N = 41) N (% Pos)	Odds ratios* (95% CI)
Bath before school			
No	12 (16.4)	2 (4.9)	1
Yes	61 (83.6)	39 (95.1)	6.3 (0.78-51.8)
Sleeping on a mattress			
No	29 (39.7)	11 (26.8)	1
Yes	44 (60.3)	30 (73.2)	2.0 (0.71-5.5)

CI, Confidence interval.
*Odds ratios were calculated with a conditional logistic regression model with the matching factors included in the model.

Discussion

Elevated total IgE levels were significantly associated with eczema (atopic dermatitis) among urban schoolchildren in Ghana, West Africa. The association was apparent despite the high proportion of elevated total IgE levels in children without eczema (70 %). Although most children with eczema showed elevated total IgE levels, there were 2 who did not show an elevated level, suggesting that not all children with eczema had an atopic constitution.

In tropical countries, house dust mites (*Dermatophagoides pteronyssinus*) have been recognized as dominant species of clinical importance, and sensitization to house dust mite allergens could be as high as 80–90% among asthmatic and allergic people.[34,35] Cockroach allergens are also associated with an increased risk of asthma and allergy in tropical countries.[36] Several allergens from house dust mites and cockroaches have been cloned and IgE cross-reactivity between the allergens of both species has been

described.[37] Elevated cockroach specific IgE levels appeared to be the driving force behind the association between elevated total IgE levels and eczema in our study.

More than half of the children with elevated total IgE levels did not show any positive skin prick tests, suggesting immune hyporesponsiveness. The association between any positive skin prick tests and eczema was less pronounced than for elevated total IgE levels and this association importantly diminished after adjustment for elevated total IgE levels. There is increasing evidence for a causal relationship between helminth infection and reduced skin prick test responsiveness to allergens.[38] Unfortunately, the protective effect of helminth infections on the development of eczema, as described in earlier studies[20] could not be addressed in this study, since only very few children in this urban population had a helminth infection at the time of the study. We cannot exclude, however, that helminth infection in the past may have induced immune hyporesponsiveness in these children. Other environmental factors or genetic factors may be alternative explanations for the apparent immune hyporesponsiveness in these children.

We observed a non-significant positive association between frequent bathing and the presence of eczema in this study. Frequent washing with soap is considered to be a healthy exercise in Ghana. Washing with soap can impair the skin barrier function by increasing the pH of the skin surface. This increased pH of the skin induces increased activity of skin proteases, resulting in premature breakdown of the corneodesmosomes. This thinning of the stratum corneum facilitates penetration of irritants and allergens inducing an immune response.[27] Despite the advice to use less soap children often continue their habits, also due to the hot, humid climate and the subsequent sweating. Nevertheless, despite the frequent use of soap, the prevalence of eczema in Ghana is still very low (around 2%).[39] Factors such as the humid climate and other environmental factors are supposedly compensating for the frequent washing habits.

There was also a weak non-significant association between sleeping on a mattress and eczema. It is possible that parents were allowing children with eczema to sleep on a mattress more often than children without eczema and this may be even more explicit among boys. Use of mattresses could also be an indicator of socio-economic status because children slept on mattresses only when these were available in the home. Sleeping on mattresses could also lead to higher exposure to house dust mite or cockroach antigens, thus influencing the occurrence of eczema.

The difficulties that we encountered when we tried to collect complete data for all children decreased the power of our study. Nevertheless we were able to collect valuable information about eczema in Ghana which should stimulate other groups to design additional studies to examine eczema in African countries.

Acknowledgements

The authors acknowledge the Director of the Institute, Prof. D. Ofori-Adjei for permission to have the study done at the Noguchi Memorial Institute for Medical Research, and Prof. M.D. Wilson, Y. Aryeetey and B. Obeng for their support. The cases were recruited from the following hospitals: Korle Bu Teaching Hospital (Prof. dr. H.A.Addo and Dr. M. Lartey), Tema General Hospital and Achimota Hospital. We thank all children, parents, teachers, fieldworkers, and laboratory workers for their participation. The study was made possible by a gift from the Gratama Foundation, the Netherlands.

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Chapter 7

Prevalence and risk factors of inflammatory acne vulgaris in rural and urban Ghanaian schoolchildren

British Journal of Dermatology 2009 **161**, pp 475-477
Published as Corresponding letter

Hogewoning AA [1,2], Koelemij I [5], Amoah AS [4], Bouwes Bavinck JN [5], Aryeetey Y [4], Hartgers F [3], Yazdanbakhsh M [3], Willemze R [5], Boakye DA [4], Lavrijsen APM [5]

[1] Dermatology, University of Ghana Medical School, Korle-Bu Teaching Hospital, Accra, Ghana,
[2] Dermatology, King Faisal Hospital, Kigali, Rwanda,
[3] Parasitology, Leiden University Medical Centre, Leiden, The Netherlands,
[4] Noguchi Memorial Institute for Medical Research, University of Ghana, Legon, Ghana,
[5] Dermatology, Leiden University Medical Centre, Leiden, The Netherlands

Abstract

Background
We aimed to investigate the prevalence and risk factors of inflammatory acne vulgaris in schoolchildren visiting rural and urban schools in Ghana.

Methods
Data on acne, age, sex, height and weight were collected from 1394 schoolchildren. The prevalence of acne was calculated for 1061 children between 9 and 16 years of age. Potential risk factors were estimated by logistic regression.

Results
In the rural schools only 1 (0.2%) out of 572 children had acne, compared to 63 (12.9 %) out of 489 children in the urban schools (P< 0.001). The prevalence of acne in the urban areas increased from 4.1% in girls and 1.3% in boys between 9 and 10 years old to 28.4% in girls and 16.4% in boys between 13 and 14 years old and leveled off in children between 15 and 16 years of age. The risk adjusted for age and BMI with 95% confidence interval of girls developing acne compared to boys was 3.2 (1.7-6.1). The risk of developing acne for children with a high BMI was 2.0 (0.9-4.3) and for children with a low BMI 0.7 (0.2-2.4) compared to children with a normal BMI.

Conclusion
The prevalence of inflammatory acne vulgaris in Ghanaian schoolchildren was very low in the rural areas and ranged between 1.3% and 28.4% in the urban areas, depending on the sex and age of the schoolchildren. Living in an urban area, increasing age, female gender and high BMI were the main risk factors associated with an increased risk of acne.

Conflict of interest
The authors state no conflicts of interest

Funding
The study was made possible by a gift from the Gratama Foundation, the Netherlands, by EU-project GLOFAL "Global view of food allergy: opportunities to study the influence of microbial exposure" FP6-2003-Food-2B contract: FOOD-CT-2005-517812, by The Netherland Organisation for Scientific Research for Global Development, WOTRO grant number WB 93-433.

Introduction

Acne vulgaris is a common skin condition in children and adolescents between the age of 10 and 18 years.[1] The prevalence of acne strongly depends on its definition and the age categories of the studied populations. In industrialized countries this condition affects between 31% and 95% of the adolescent population.[1-4] Acne can also persist beyond adolescence.[5]

The prevalence of acne is considerably lower in developing countries.[6-9] Community-based studies, specifically studying acne vulgaris in African countries are scarce and are summarized together with studies performed in other developing countries in Table 1. Hospital-based studies on skin diseases, among which acne, in a population of people of all ages were more frequently reported (Table 1). The prevalence of acne in hospital-based studies in Africa ranged between 2.8% and 8.9% with the exception of a hospital-based study performed in South Africa which showed a prevalence of 16%.[10-17] Only few studies compared the prevalence of acne in schoolchildren who are living in rural and urban areas (Table 1). One study performed in Ethiopia showed a higher prevalence of acne in rural areas (3.3%) compared to urban areas (0.8%).[18] Another study performed in Brazil, however, found the opposite with a prevalence of acne in the rural areas ranging between 0% and 0.7% compared to prevalences ranging between 0.7% and 3.5% in the urban areas.[8]

Different prevalences of acne between industrialized and developing countries may be explained by differences in genetic and environmental risk factors such as diet, usage of medication or cosmetics, and body weight, reflecting the nutritional status of the children. The difference in acne prevalence between rural and urban areas within one country, however, cannot easily be explained by genetic factors and is probably influenced by environmental differences or different food habits.

The purpose of this study was to estimate the prevalence of inflammatory acne vulgaris in schoolchildren living in rural and urban areas of the Greater Accra Region of Ghana and to study risk factors of inflammatory acne vulgaris.

Methods

Recruitment of schoolchildren and Ethical Approval

This cross-sectional study is part of a larger project in which the association of helminth infection and other potential risk factors with allergic sensitization and atopic eczema among school children in Ghana are studied.[19] The children were recruited from 13 schools between January 2006 and March 2007. Between 5 and 16 February 2007 two dermatologists (A.P.M. Lavrijsen and A.A. Hogewoning) visited 11 of these schools in rural and urban areas in the Greater Accra Region of Ghana and screened a total of 1394

Table 1 Reported prevalences of acne vulgaris in developing countries.

Publication year	Country and reference	Study population (N)	Age group (years)	Prevalence of acne	Rural	Urban	Study design*	Study aim**
Africa								
1997	Ethiopia[18]	219	5-15	1.8%	3.3%	0.8%	C	SD
1998	Mali[13]	10575		4.2%			H	SD
2000	Ethiopia[17]	1000	0-12	8.9%			H	SD
2001	Ghana and UK[12]	2254 3383		4.6%[&] 5.5%[&]			H	SD
2003	Egypt[40]	8008		5.4%			C	SD
2003	South Africa[11]	7029		16%			H	SD
2004	Nigeria[16]	1091		2.8%			H	SD
2005	Nigeria[15]	2871	18-73	4.3%			H	SD
2007	Nigeria[14]	5982	0-92	6.7%			H	SD
2007	Senegal[10]	93	14-46	5.3%			H	A
2008	Ghana (this study)	1061	9-16	6.0%	0.2%	12.9%	C	A
Asia and South America								
1981	Brazil[8]	9955	6-16	2.7%	0-0.7%	0.2-3.5%	C	SD
1998	Peru[24]	2214	12-18	42%			C	A
2002	Papua New Guinea, Paraguay[6]	1315		0%			C	A
2003	India[9]	12586	6-14	0.93%			C	SD

*C = Community-based, H = Hospital-based; **SD = Prevalence of skin disease, A = Prevalence of acne; [&]acne and rosacea pooled.

schoolchildren for inflammatory acne vulgaris, atopic eczema and other skin diseases. A questionnaire was administered to each child, collecting information concerning living conditions. The skin of each child was fully examined by one or both dermatologists. All skin diseases observed, with special attention for inflammatory acne vulgaris and atopic eczema were recorded on the questionnaire.

Children whose parents consented by signing or thumb printing an informed consent form were enrolled in the study. The Institutional Review Board of the Noguchi Memorial Institute for Medical Research in Ghana granted ethical approval for this study.

Definition of acne

The clinical diagnosis of inflammatory acne vulgaris, ascertained by two dermatologists experienced in dermatology of African skin, was used for recording the presence of inflammatory acne. The diagnosis of inflammatory acne was defined by the presence of at least 6 pustules or papulopustules on the face.[20] The severity grade was not assessed separately.

BMI

The height and weight of the schoolchildren were measured to calculate the Body Mass Index (BMI) as a marker of nutritional status. The measurements of height and weight were done in a period ranging between 12 months before and 1 month after the dermatological examination. The BMI was calculated by dividing the body weight with the square of height (kg/m^2).

The BMI in childhood is strongly dependent on age and to a lower extent on sex.[21] Two recent studies of Cole et al. have provided cut-off points for BMI in childhood that are based on international data.[21,22] The cut-off point for overweight was linked to a BMI of > 25 kg/ m^2 (high BMI) and for underweight linked to a BMI of < 17 kg/ m^2 at age 18 years (low BMI).

Statistical Analysis

The differences in prevalence (point prevalence) of inflammatory acne vulgaris, BMI and age in relation to the four types of schools were analyzed with the Pearson's chi square test and ANOVA test. Comparison of age, sex, height, weight and BMI between children with and without inflammatory acne was made with the Pearson's chi square test and an independent T-test.

Logistic regression analyses were performed taking the presence or absence of inflammatory acne as the dependent variable. The model included gender, age and BMI. The odds ratios, 95% confidential intervals and, where appropriate, p-values were reported. In all cases, statistical tests were considered significant when p-values were less than 0.05. All statistical analyses were performed using the SPSS 16.0 software package.

Study Area and Socio-economic Status

The general study area was in the Greater Accra Region of Ghana between longitudes W 000.35377° and E 000.42752° and latitudes N 005.72647° and N 005.53550°. All the urban schools were in the Accra Metropolis where we included two private rich schools with a high socio-economic status (Mona Lisa School where 36 and Morning Star where 35 children were seen); two private schools with middle to high socio-economic status (Greenhill International where 189 and The Youngster International where 167 children were seen) and one public school with low socio-economic status (Immanuel Presbyterian Primary School where 214 children were seen). Five rural schools were located in Dangme East (Agbedrafor where 119, Anyamam where 214, Goi where 79, Koluedor R/C Primary School where 128 and Toflokpo where 108 children were seen) and one rural school was located in Ga East (Pantang Primary and JSS where 105 children were seen). Main income earning activity in the Ga district is farming, while fishing, salt mining and farming are characteristics of Dangme East.

Results

A total of 1394 children participated in the study (Table 3). No statistically significant difference in gender distribution among the four types of schools was found (Table 3). The children in the rural schools were, however, significantly shorter and weighted less than those in the urban schools, except for the 4-8 year old children in the urban public schools. This difference was also reflected in significant differences in median BMI in older children between the schools. The children in the urban private rich schools had a higher median BMI compared with the other types of school, which was also reflected by a higher percentage of children with a high age-adjusted BMI in the urban private rich schools (Table 3).

Under the age of 9 years we did not observe inflammatory acne vulgaris in any of the schoolchildren. Children in this age category were therefore excluded from further analysis. Children in the age category between 17 and 20 years were also excluded from further analyses since there were only 13 children in this age group (Table 3).

In the rural schools only 1 (0.2%) out of 572 children had acne, compared to 63 (12.9 %) out of 489 children in the urban schools (P< 0.001). Figure 1 shows the distribution of inflammatory acne vulgaris in the urban schools amongst 9 to 16 year old boys and girls. The prevalence of acne in the urban schools increased from 4.1% in girls and 1.3% in boys between 9 and 10 years old to 28.4% in girls and 16.4% in boys between 13 and 14 years old and leveled off in children between 15 and 16 years of age to 18.8 % in girls and 13.3% in boys (Figure 1). This confirms that girls develop acne at a younger age than boys. At the age of 15 years the prevalence of inflammatory acne among boys appeared to approach the prevalence among girls.

Table 2 BMI cut-off points for low, normal and high BMI in relation to age which were used in our study.

	Girls				Boys			
Age	Thinness grade 2	Thinness grade 1*	Normal	High**	Thinness grade 2	Thinness grade 1*	Normal	High**
5	< 12.99	12.99-13.85	13.86-17.19	17.20 and more	< 13.22	13.22-14.12	14.13-17.44	17.45 and more
6	< 12.90	12.90-13.81	13.82-17.52	17.53 and more	< 13.10	13.10-14.03	14.04-17.70	17.71 and more
7	< 12.95	12.95-13.92	13.93-18.02	18.03 and more	< 13.09	13.09-14.07	14.08-18.15	18.16 and more
8	< 13.08	13.08-14.13	14.14-18.68	18.69 and more	< 13.17	13.17-14.23	14.24-18.75	18.76 and more
9	< 13.29	13.29-14.42	14.43-19.44	19.45 and more	< 13.34	13.34-14.48	14.49-19.45	19.46 and more
10	< 13.59	13.59-14.80	14.81-20.19	20.20 and more	< 13.58	13.58-14.79	14.80-20.19	20.20 and more
11	< 14.01	14.01-15.31	15.32-21.19	21.20 and more	< 13.87	13.87-15.15	15.16-20.88	20.89 and more
12	< 14.56	14.56-15.92	15.93-22.13	22.14 and more	< 14.25	14.25-15.57	15.58-21.55	21.56 and more
13	< 15.14	15.14-16.56	16.57-22.97	22.98 and more	< 14.74	14.74-16.11	16.12-22.26	22.27 and more
14	< 15.72	15.72-17.17	17.18-23.65	23.66 and more	< 15.28	15.28-16.68	16.69-22.95	22.96 and more
15	< 16.22	16.22-17.68	17.69-24.16	24.17 and more	< 15.82	15.82-17.25	17.26-23.59	23.60 and more
16	< 16.62	16.62-18.08	18.09-24.53	24.54 and more	< 16.34	16.34-17.79	17.80-24.18	24.19 and more
17	< 16.89	16.89-18.37	18.38-24.83	24.84 and more	< 16.80	16.80-18.27	18.28-24.72	24.73 and more
18 and older	< 17.00	17.00-18.49	18.50-24.99	25.00 and more	< 17.00	17.00-18.49	18.50-24.99	25.00 and more

*Adapted from [21]; **Adapted from [22]

Table 3 Baseline characteristics of the study population.

		Type of school				
	Whole group	Rural	Urban public	Urban private	Urban private rich	P-value
No of children	1394	753	214	356	71	
Male: N (%)	660 (47.3)	361 (47.9)	103 (48.1)	161 (45.2)	35 (49.3)	P=0.823*
Age						
Median (SD)	11.0 (2.4)	10.0 (2.3)	11.0 (2.7)	10.5 (2.4)	11.0 (2.6)	P=0.040**
Age: N (%)						
4- 8	317 (22.8)	169 (22.5)	45 (21.0)	90 (25.3)	13 (18.3)	P<0.001*
9-12	802 (57.7)	473 (63.1)	105 (49.1)	186 (52.2)	38 (53.5)	
13-16	259 (18.6)	99 (13.2)	62 (29.0)	79 (22.2)	19 (26.8)	
17-20	13 (0.9)	9 (1.2)	2 (0.9)	1 (0.3)	1 (1.4)	
Height (cm) Median (SD)						
4- 8	121.7 (7.6)	120.5 (7.2)	118.6 (7.9)	125.5 (7.1)	128.3 (8.9)	P<0.001**
9-12	136.6 (9.7)	134.0 (8.8)	138.5 (7.9)	142.1 (10.1)	143.1 (9.1)	P<0.001**
13-16	152.3 (9.8)	145.8 (9.2)	153.8 (7.4)	156.1 (8.1)	158.4 (8.6)	P<0.001**
17-20	155.1 (10.3)	154.1 (11.6)	162.8 (1.1)	156.3 (0)	154.4 (0)	***
Weight (kg) Median (SD)						
4- 8	24.0 (4.8)	24.0 (4.5)	21.0 (3.8)	24.0 (4.6)	27.0 (8.2)	P=0.001**
9-12	32.0 (8.5)	32.0 (6.5)	32.0 (7.6)	32.0 (11.8)	36.0 (9.9)	P<0.001**
13-16	43.0 (11.4)	39.0 (7.7)	46.0 (9.6)	45.0 (12.4)	52.0 (16.3)	P<0.001**
17-20	49.0 (10.2)	45.0 (9.2)	50.0 (2.8)	66.0 (0)	56.0 (0)	***
BMI (kg/m^2) Median (SD)						
4- 8	16.0 (2.1)	16.6 (2.1)	14.5 (1.7)	15.2 (2.0)	16.4 (3.7)	P<0.001**
9-12	17.2 (2.7)	17.6 (2.1)	16.4 (2.5)	16.2 (3.6)	17.0 (3.3)	P=0.006**
13-16	18.6 (3.5)	18.3 (2.3)	19.4 (3.5)	17.8 (4.0)	20.0 (5.0)	P=0.007**
17-20	19.6 (3.0)	18.8 (1.7)	18.9 (1.3)	27.0 (0)	23.5 (0)	***
BMI, age adjusted: N (%)[£]						
Thinness grade 2	72 (5.2)	26 (3.5)	17 (8.1)	23 (6.5)	6 (8.5)	P=0.000*
Thinness grade 1	190 (13.8)	71 (9.6)	41 (19.4)	74 (20.9)	4 (5.6)	
Normal	999 (72.7)	587 (79.4)	140 (66.4)	223 (63.0)	49 (69.0)	
High	114 (8.3)	55 (7.4)	13 (6.2)	34 (9.6)	12 (16.9)	
Missing information	19	14	3	2	0	

BMI, body mass index

* Chi-square test; **ANOVA test; [£] Thinness grade: See for definition Table 2.

***No statistics performed, because of low numbers of children in some categories.

Since there was only one child with acne in the rural schools and since rural schools significantly differed from urban schools with respect to length, weight and BMI, the analyses of risk factors for acne were restricted to the 9-16 year-old children attending the urban schools (Table 4). Children with inflammatory acne were significantly older and more often girls compared to children without acne. They more often showed a high BMI in comparison with children without inflammatory acne (Table 4). The percentage of acne was not significantly different between the public, private and private rich urban schools (Table 4). Female gender, increasing age and high BMI appeared to be independent risk factors for inflammatory acne vulgaris after adjustment for these factors (Table 4).

Table 4 Risk factors for inflammatory acne vulgaris in 9 to 16 year old children attending urban schools.

	No Acne N (%)	Acne N (%)	Adjusted odds ratio (95% CI)*
Sex:			
Male	206 (93,6)	14 (6,4)	1
Female	220 (81,8)	49 (18,2)	3.2 (1.7-6.1)
Age:			
9-12	301 (91.5)	28 (8.5)	1
13-14	99 (76.7)	30 (23.3)	3.3 (1.8-5.9)
15-16	26 (83.9)	5 (16.1)	2.6 (0.87-7.5)
BMI**			
Thinness grade 2 and 1	31 (91,2)	3 (8,8)	0.68 (0.19-2.4)
Normal	356 (88,1)	48 (11,9)	1
High	36 (75,0)	12 (25,0)	2.0 (0.93-4.3)
Urban School			
Public	149 (89.2)	18 (10.8)	1
Private	226 (85.3)	39 (14.7)	1.5 (0.82-2.9)
Private rich	51 (89.5)	6 (10.5)	0.94 (0.34-2.6)

*The odds ratios are adjusted for sex age, BMI and type of school.
**This information is missing from 3 children without acne.

Discussion

The prevalence of inflammatory acne vulgaris in Ghana was less than 1% in the rural schools and varied between 1.3% and 28.4% in the urban schools depending on the age and sex of the schoolchildren. The prevalence of acne in Ghana was low in comparison with some industrialized countries[1-4] but was higher in the urban schools compared to other community-based studies of schoolchildren in developing countries like India, Ethiopia and Brazil (Table 1).[8;9;18] Although the diagnoses of acne in the latter studies were not clearly defined, these studies support our finding that the prevalence of acne is much lower in developing countries than in industrialized countries.

With the changing socio-economic situation in developing countries, especially westernization in urban areas, it is believed that the prevalence of acne vulgaris in developing countries will increase to the level of industrialized countries. This has already been observed among the Inuit people in Canada who had given up nomadic life and had moved into settlements adopting a western lifestyle. Acne vulgaris has become common among the Inuit, although the condition used to be rare among this ethnic group.[23]

In our study girls appeared to develop acne at a younger age than boys, but boys were catching up as they became older (Figure 1). Between the ages of 16 and 18 boys are more likely to have acne.[1;24;25]

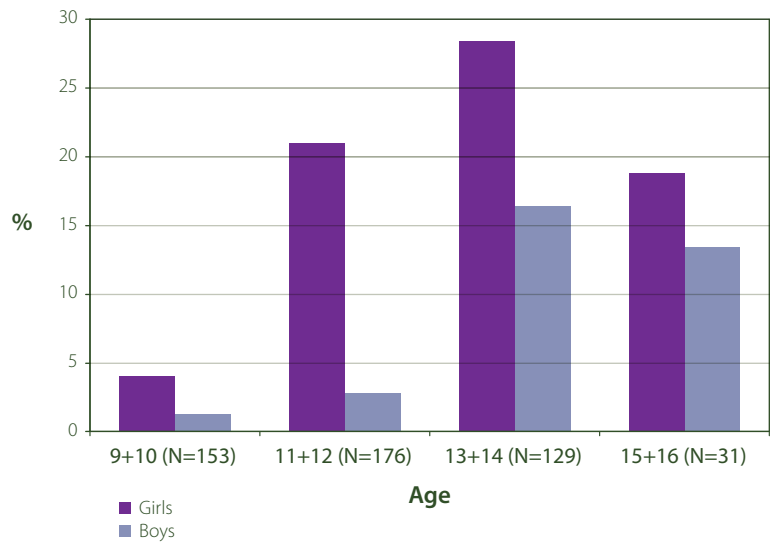
The striking difference between the prevalences of acne in rural and urban schools may reflect differences in genetic background or differences in environmental exposure. It is not likely that genetic factors explain the different prevalences of acne between rural and urban schools in our study. The most important environmental differences between the rural and urban areas in the greater Accra region are the lower food intake and the higher exposure to helminth infections in the rural areas, which are factors that both can lead to a lower nutritional status of the children. The significantly lower height and weight of the rural children may reflect chronic malnutrition in the rural areas.[26]

A lower nutritional status may protect against the development of acne. The observed association between a higher BMI and acne among the children who were attending the urban schools would nicely fit in this hypothesis. An association between overweight and acne has also been described by Tsai et al. [27] and Bourne. [28] However, Bourne et al. found this association only in adult males.[28]

The observed differences in height and BMI reflecting the nutritional status of the children may not be the only factors explaining the profound difference in the prevalence of acne between the rural and urban schools. Other factors which could explain this difference may be the use of cosmetics or the consumption of different types of food.

In urban areas cosmetics with comedogenic ingredients, bleaching creams containing corticosteroids and pomade are much more available and potentially could contribute to the higher prevalence of acne in urban areas.

Figure 1 Percentage of children with acne in urban areas between 9 and 16 years of age.



An association between diet and acne has long been suggested.[29] This has also been suggested by Cordain et al.[6] who found no cases of acne among non-Westernized societies like the Kitavan Islanders of Papua New Guinea and Ache hunter gatherers of Paraguay. They postulated that the difference in the prevalence of acne between developing and industrialized countries cannot be solely attributed to genetic differences but likely results from different environmental factors such as diet, which have a substantially lower glycemic index than Western diets.[6]

Evidence for the relation between diets with high glycemic index and acne is gradually accumulating.[29] Insulin-like growth factor 1 (IGF-1) and high levels of insulin, induced by a high glycemic-load diet, stimulate sebaceous glands and the synthesis of androgens.[6;29-33] A recent randomized controlled trial using a high-protein, low glycemic-load diet versus a high glycemic-load diet showed a decrease in acne severity and improved insulin sensitivity.[32;33] It is likely that rural children in our study may have had limited access to processed products and products with refined carbohydrates and this may be one of the possible reasons to explain the difference in acne prevalence in urban and rural children.[34-37] Consumption of milk, containing hormonal constituents, is also described to contribute to the formation of acne.[29;38;39]

Additional community-based studies in developing countries are necessary, especially studies comparing rural with urban areas to confirm the profound difference in the prevalence of acne between these areas.

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Chapter 8

Skin diseases among children in Africa

Arjan Hogewoning, Sjan Lavrijsen, Colette van Hees

This chapter of the thesis reflects the wish of putting acquired knowledge and information into a format which is useful in daily practice in Africa. It describes the epidemiology, etiology and pathogenesis, clinical symptoms and the management and treatment of common and typically tropical skin diseases among children in Africa. The described management of the diseases is experience based. There is little evidence based pharmacotherapy in children.
This chapter is complementary to the book 'Common Skin Diseases in Africa. An illustrated Guide' by Colette van Hees and Ben Naafs (ISBN/EAN: 978-90-808016-2). There is some overlap in text and illustrations. However "Skin diseases among children in Africa" focuses specifically on children, and, in line with the rest of the thesis, on epidemiology. Illustrations were provided by Arjan Hogewoning, Sjan Lavrijsen, Colette van Hees, Ben Naafs, Johan van der Stek and Rosemarie Moser.

This chapter is meant to be a practical guide for general practitioners, health care workers, students and all others who are working in the medical field. The list of skin diseases described is far from complete and will benefit from continuous improvements and additions. Modern communication tools like websites provide these functionalities. We created a freely accessible website named www.africanskindiseases.org to cater for this. "Skin diseases among children in Africa" and "Common skin diseases in Africa" are the first of hopefully many publications accessible through this website.

Skin diseases among children in Africa

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Skin infections

BACTERIAL

Pyoderma
(Impetigo, ecthyma, folliculitis, furuncle)

The majority of the skin diseases found among schoolchildren in Africa are dominated by fungal infections and pyoderma.[1-4] Factors like overcrowding, malnutrition and climatic conditions such as heat and humidity lead to an increase in bacterial infections in tropical and semi-tropical countries.[5;6]

The term pyoderma is used to describe bacterial skin infections; ***impetigo***, ***ecthyma***, ***folliculitis***, ***furuncle*** or ***carbuncle***. It is usually caused by *staphylococci* and/ or *pyogenic streptococci* which may penetrate the skin primarily or secondary to trauma or other infections. Invasive infections may spread from superficial infections or enter through a defect in the skin such as interdigital tinea pedis.[7;8] A problem is the misuse of antibiotics available without prescriptions.

Reasons for concern are recent reports of growing incidences of *S.aureus* bacteraemia coupled with high prevalences of methicillin resistance (MRSA), particularly in HIV-infected children.[9] This growing rate of resistance to currently recommended antibiotics for skin and soft tissue infections could pose a significant health threat in sub-Sahara Africa, especially in regions with limited access to microbiological laboratory facilities and to adequate antimicrobial agents.[10;11]

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Impetigo

Epidemiology

Impetigo is a frequently observed superficial, very contagious, bacterial infection which can be divided in a non-bullous and a bullous form. Non-bullous impetigo accounts for more than 70% of cases of impetigo. It is frequently diagnosed in regions with a warm humid climate. Overcrowding, malnutrition and lack of hygiene also play an important role.

Etiology and pathogenesis

The predominant cause of non-bullous impetigo is *Staphylococcus aureus* although also *Streptococcus pyogenes* can be involved, especially in tropical countries. Bullous impetigo is nearly always caused by a coagulase positive *S. aureus*. These bacteria belong to a specific group (phage group 2) which produces an exfoliative toxin responsible for the blister formation. Phage group 2 *S. aureus* are also responsible for the development of the staphylococcal scalded skin syndrome (SSSS) which occurs mainly in neonates and infants.

Clinical findings

Impetigo usually occurs on exposed areas like the face and extremities. Non-bullous impetigo starts often with a pustule which can develop rapidly and lead to the formation of yellow or brown colored crusts. Usually there is no pain but the lesions may be itchy. In the majority of cases regional lymphadenopathy can be found. Bullous impetigo presents with large blisters which rupture easily. They are usually localized on the face, extremities and the diaper area and they heal without scarring.

Differential diagnosis

- Herpes simplex
- Varicella
- Candidiasis
- Insect bites (hypersensitivity response)
- Pemphigus
- Trauma (thermal)

Management

- Impetigo is highly contagious, spreading needs to be prevented. Do not share the same towels and change clothes and towels frequently.
- In limited cases local therapy is usually sufficient. Wash with betadine shampoo daily and apply gentian violet paint 0.5%, mupirocin ointment, fusidic acid cream, sulphur 5% in zinc oxide cream or betadine ointment twice daily on the lesions.
- In moderate /severe cases an oral antibiotic, active against both *streptococci* and *staphylococci* (also beta-lactamase producing strains) like dicloxacillin is the drug of first choice. In case of penicillin-allergic patients, erythromycin can be given. When MRSA is suspected, cefalexin is an option.

* **Flucloxacillin** (British National Formulary)

- Child under 2 years: quarter of the adult dose: 62.5-125 mg every 6 hours.
  Oral solution (Syrup, flucloxacillin, 25 mg/1mL) 2.5 mL-5 mL 4 times daily.
- Child 2-10 years : half of the adult dose: 125mg-250mg every 6 hours.
  Oral solution (Syrup, flucloxacillin, 25 mg/mL) 5 mL 4 times daily or
  Capsules (flucloxacillin, 250 mg) 1 capsule 4 times daily.
- Child above 10 years: adult dose: 250-500 mg 4 times daily.
  Capsules (flucloxacillin, 250 mg or 500 mg) 1 capsule 4 times daily.

* **Erythromycin** (British National Formulary)

- Child up to 2 years: 125 mg 4 times daily.
  Oral solution (Syrup, erythromycin, **25** mg/1mL) 5mL 4 times daily.
- Child 2-8 years: 250 mg 4 times daily.
  Oral solution (Syrup, **50** mg/1mL) 5 mL 4 times daily or
  Capsules (erythromycin, 250mg) 1 capsule 4 times daily.
- Child above 8 years: adult dose: 250-500 mg 4 times daily.
  Capsules (erythromycin, 250 mg or 500 mg) 1 capsule 4 times daily.

* **Cefalexin** (British National Formulary)

- Child under 1 year: 125 mg every 12 hours.
  Oral solution (Syrup, cefalexin **25** mg/1mL) 5 mL 2 times daily.
- Child 1-5 years: 125 mg every 8 hours.
  Oral solution (Syrup, cefalexin 25 mg/1mL) 5 mL 3 times daily.
- Child 6-12 years: 250 mg every 8 hours.
  Oral solution (Syrup, cefalexin **50** mg/1mL) 5 mL 3 times daily or
  Capsules (cephalexin 250 mg) 1 capsule 3 times daily.
- Above 12 years: adult dose: 250 mg every 6 hours or 500mg every 12 hours.
  Capsules (cefalexin 250 mg) 1 capsule 4 times daily or 2 capsules 2 times daily or 1 capsule (cefalexin 500mg) 2 times daily.

Clinical pictures

Non-bullous impetigo: Small erosions with brown-yellow crusts

Bullous impetigo: Superficial blisters/ erosions

Reference list see Pyoderma

Bacterial folliculitis

Epidemiology

Bacterial folliculitis is mainly diagnosed among children and caused by *S.aureus*.

Etiology and pathogenesis

Folliculitis is an inflammation of hair follicles which, when bacterial, is usually caused by *staphylococci*. Sometimes also other causative agents like *streptococci* or *Pseudomonas aeruginosa* are involved. Minor trauma caused by scratching, physical or chemical injury and the use of topical steroids can induce folliculitis.

Clinical findings

Follicular dome shaped yellow papulopustules are surrounded by a red areola. Lesions develop in crops; the most affected areas are the scalp, thighs and buttocks.

Differential diagnosis

- Insect bites
- *Pityrosporum* folliculitis
- Acne vulgaris
- Folliculitis caused by oily or tar products
- Follicular pustules can also occur in or around a mycotic infection

Management

- Local therapy is usually sufficient. As local treatment wash with betadine shampoo daily and apply mupirocin ointment, fusidic acid cream, sulphur 5% in zinc oxide cream, gentian violet paint 0.5% or betadine ointment twice daily on the lesions.
- Avoid oil and vaseline based topical products.
- Severe or recurrent infections may be treated systemically with oral antibiotics. like flucloxacillin. In case of penicillin-allergic patients, erythromycin can be given. For the dosages see impetigo.

Reference list see Pyoderma

Clinical picture

Folliculitis on a leg

Ecthyma

Epidemiology

Ecthyma describes deeper punched out lesions which can be complicated by lymphangitis and cellulitis. Overcrowding, poor hygiene and malnutrition are important factors in its development. Ecthyma often occurs as a secondary lesion after scratching itchy lesions such as insect bites or after local trauma.

Etiology and pathogenesis

Ecthyma is usually caused by *Streptococcus pyogenes* but may be caused by *Staphylococcus aureus* as well. It occurs mostly on the legs where infection extends into the sub-cutaneous tissue.

Clinical findings

The initial lesion is a blister, surrounded by redness and edema. In the beginning it can resemble impetigo but ecthyma extends into the sub-cutaneous tissue and causes a painful ulcer. It usually heals with the formation of scars.

Differential diagnosis

- Impetigo
- Burns
- Ecthyma gangrenosum (caused by *Pseudomonas aeruginosa)*. This usually occurs in patients with immunedeficiency
- Anthrax

Management

- Removal of the crust.
- As local treatment wash with betadine shampoo daily and apply Gentian violet paint, mupirocin ointment, fusidic acid cream, sulphur 5% in zinc oxide cream or betadine ointment twice daily on the lesions.
- A small spectrum antibiotic therapy against *Strep.pyogenes* and *Staph.aureus* is recommended. Phenoxymethylpenicillin can be given. In case of penicillin-allergic patients, erythromycin is a good alternative. For the dosage see impetigo.

Clinical picture

Painful ulcer on the leg

*__Phenoxymethylpenicillin__ (British National Formulary)

- Child up to 1 year: 62.5 mg 4 times daily.
  Oral solution (Syrup, phenoxymethylpenicillin, 25 mg/1mL)
  2.5 mL 4 times daily.
- Child 1- 5 years: 125 mg 4 times daily.
  Oral solution (Syrup, phenoxymethylpenicillin, 25 mg/1mL) 5mL 4 times daily.
- Child 6-12 years: 250 mg 4 times daily.
  Tablets (phenoxymethylpenicillin, 250 mg) 1 tablet 4 times daily.
- Above 12 years: 500 mg 4 times daily.
  Tablets (phenoxymethylpenicillin, 250 mg) 2 tablets 4 times daily.

Reference list see Pyoderma

Furuncle

Epidemiology

A furuncle is a painful abscess around the hair shaft and in the perifollicular skin. Furuncles are more common in boys than in girls.

Etiology and pathogenesis

Furuncles occur in hair-bearing skin. The causative agent is nearly always *Staphylococcus aureus*. Risk factors for the development of furuncles are: a humid environment, obesity or malnutrition, HIV infection and *S. aureus* carriage.

Clinical findings

A furuncle presents as a painful, deep-seated well circumscribed papulopustule which develops into a nodule with central necrosis and pus. Sites of predilection are: the neck, buttocks, groin and armpits. When there is a group of furuncles which form one nodular lesion with multiple drainage points it is called a carbuncle.

Differential diagnosis

- Hidradenitis suppurativa
- Folliculitis
- Acne vulgaris
- Sinus pilonidalis
- Myiasis

Management

- Frequent application of a moist compress to stimulate drainage.
- Do not share the same towels and change clothes and towels frequently.
- In uncomplicated lesions local therapy is usually sufficient. As local treatment wash with betadine shampoo daily and apply fusidic acid cream, sulphur 5% in zinc oxide cream or betadine ointment twice daily on the lesions.
- Lesions with surrounding cellulitis, or furuncles located on the face demand systemic antibiotic treatment. Flucloxacillin (which is active against beta-lactamase producing strains) is the drug of first choice. In case of penicillinallergic-patients, erythromycin or cefalexin can be given. For dosages see impetigo.
- When furuncles recur and S. aureus carriage is suspected, the patient can be treated with mupirocin nasal ointment, to apply three times daily to the inner surface of each nostril for the first 5 days of each month. In poor resource countries the use of gentian violet paint 0.5% is an option.

Clinical pictures

Caucasian boy 11 years, some furuncles on the adomen

Furuncle, detail

Reference list see Pyoderma

Buruli ulcer

Epidemiology

Buruli ulcer is caused by Mycobacterium ulcerans. It is the third most common mycobacterial disease among humans, after tuberculosis and leprosy. The incidence is highest in children up to 15 years old. Among the younger children males are more infected. Buruli ulcer is endemic in Africa and most patients live in West Africa. In Ghana seasonal variation has been described.[1-3] Environmental factors like deforestation, increased manual agriculture of wetlands, illegal diamond or gold digging etcetera seem to play an important role.[3-7]

Etiology and pathogenesis

Most probably the mode of transmission is by skin trauma at sites contaminated by *M. ulcerans*. The pathway of transmission remains unknown, despite many years of research. The primary risk factor associated with Buruli ulcer is proximity to slow moving water and direct water contact. In arid regions Buruli ulcer is usually absent. *M.ulcerans* contains a plasmid that produces a diffusible necrotizing toxin in tissues, mycolactone, which gives the ulcers the typical undermining aspect.

Clinical findings

Buruli ulcer is a necrotizing skin disease that can leave patients with prominent scars and lifelong disability. After infection a painless nodule is formed which eventually ulcerates. This process evolves very slowly, and large body areas may eventually be affected. Despite their impressive appearance the lesions are strikingly painless and patients are usually otherwise healthy. There are several types of lesions:

- I Small early lesion (eg, nodules, papules, plaques, ulcers < 5 cm in diameter)
- II Non ulcerative and ulcerative plaque and edematous forms
- III Large ulcerative lesions (>5 cm in diameter)

Besides the skin and the subcutis deeper structures may be affected, leading to osteomyelitis and bone destruction. [8-10] [11]

Differential diagnosis

- Other tropical ulcers[12]
- Leishmaniasis
- Cutaneous tuberculosis
- Onchocerciasis nodules
- Fungal skin infections.

Management

- In the recent past excision was the treatment of choice but now serves more as an adjunct to antibiotic treatment.[13-15]
- A combination of rifampicin and streptomycin for 8 weeks should be given. Rifampicin, 10 mg/kg body weight by mouth daily for 8 weeks and streptomycin, 15 mg/kg body weight by intramuscular injection daily for 8 weeks. Because its side effects (ototoxicity and nephrotoxicity) streptomycin is more and more replaced by clarithromycine, ciprofloxacin, moxifloxacin or amikasin.
- If surgery is combined with antibiotic therapy only minimal surgery to excise necrotic tissue is required when antibiotics have arrested progression of the disease.
- Interventions to minimize or prevent disabilities.
- BCG Vaccination programmes, though the protective effect is short-term and according to some studies non existing[13]
- The treatment depends on the different clinical categories.

* Category I (small early lesion), if possible a direct excision and suturing is recommended. Antibiotics should be started at least 24 hours before surgery and continue for 4 weeks. If surgery is not possible all lesions in this category can be treated with antibiotics for 8 weeks. Category I can be treated in smaller clinics / primary health care centers and referral hospitals.

* Categories II and III should be treated with antibiotics for at least 4 weeks, then surgery (if necessary), followed by another 4 weeks of antibiotics. Both categories should be treated in a district or tertiary health care facility. (see: http://www.who.int/buruli/information/antibiotics/en/.)

Clinical pictures

Ghanaian boy 12 year, ulcerating plaque

Ulcerative lesion on the foot with typical "undermining"

Clinical picture

Ghanaian boy, 12 years old
Large ulcerative lesion with undermining

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Leprosy

Epidemiology

The newly detected number of patients (NCD) with leprosy in 2010 was 228.474, which is about 50% of the NCD in 1985. Up to 10% of new leprosy cases occur in children under 15 years.[1;2] This means that even though elimination strategies have had a positive effect, leprosy is still endemic in South East Asia, South America and Africa, India and Brazil being the most affected. An explanation may be that contagious patients are not discovered in time.

Etiology and pathogenesis[3]

Leprosy is an infectious and immunological disease caused by *Mycobacterium leprae*. It is transmitted by leprosy patients who may carry many bacilli, particularly multibacillary patients, usually by sneezing or coughing. Of those infected only few develop leprosy. Leprosy is a generalized disease which especially affects skin and nerves. The clinical presentation and damage done depend on host immunity. Skin and nerve involvement and damage may occur by infiltration with *M. leprae,* or in particular during leprosy reactions, which may occur before, during or after treatment.

Clinical findings[4-6]

In paucibacillary (PB) leprosy there is strong cellular immunity. Five or less well demarcated hypopigmented or slightly erythematous skin patches with loss of sensation are seen on the skin and no bacilli are found in the patches. One or more local or regional nerves may be enlarged. In multibacillary (MB) leprosy there are more than five skin lesions which may be flat, popular, nodular or plaques. In total absence of a cell mediated immune response the whole skin may be infiltrated (Lepra bonita). MB patients have positive skin smears and are contagious.

Leprosy reactions may cause severe nerve damage if not recognized and treated properly. Symptoms of reversal reactions (RR) are erythema and swelling of previous lesions, appearance of new lesions or enlargement, tenderness and loss of function of nerves.[7] Sometimes there is acral edema. In erythema nodosum leprosum (ENL) tender erythematous nodules appear, nerves may become tender and the patient usually feels sick. Other organs may be affected too, causing for example arthritis, lymphadenitis, orchitis and iridocyclitis. Ulceration is secondary to the loss of protective sensation and may lead to cellulitis, deep infections, osteomyelitis and consequently loss of digits, causing deformity.

Differential diagnosis

- Tinea corporis
- Lupus vulgaris
- Atypical mycobacterial infection
- Leishmaniasis

- Neurofibromatosis
- Sarcoidosis
- Pityriasis versicolor
- Granuloma annulare
- Vitiligo
- Erythema nododum
- Yaws
- Kaposi sarcoma

Management of uncomplicated leprosy as advised by the WHO

- PB leprosy, children under 10 years: Rifampicine 300 mg once a month under supervision plus dapsone 25 mg daily unsupervised for 6 months (sometimes 12 months treatment may be needed), 6 monthly doses in 9 months are considered enough.
- PB leprosy, children 10-14 years: Rifampicine 450 mg once a month under supervison plus dapsone 50 mg daily unsupervised for 6 months (sometimes 12 months treatment may be needed), 6 monthly doses in 9 months are considered enough.
- PB leprosy above 14 years: Rifampicine 600 mg under supervison plus dapsone 100 mg daily unsupervised for 6 months (sometimes 12 months treatment may be needed), 6 monthly doses in 9 months are considered enough.
- MB leprosy children under 10 years: Rifampicine 300 mg and clofazimine (lampren) 100 mg under supervision monthly plus dapsone 25 mg daily and clofazimine 50 mg twice a week unsupervised for 12 months. (sometimes 24 months treatment may be needed), 12 monthly doses in 18 months are considered enough.
- MB leprosy children 10-14 years: Rifampicine 450 mg and clofazimine (lampren) 150 mg under supervision plus dapsone 50 mg daily and clofazimine 50 mg every other day unsupervised for 12 months. (sometimes 24 months treatment may be needed), 12 monthly doses in 18 months are considered enough.
- MB leprosy above 14 years (50-80 kg): Rifampicine 600 mg and clofazimine (lampren) 300 mg plus dapsone 100 mg plus dapsone 100 mg daily and clofazimine 50 mg daily unsupervised for 12 months (sometimes 24 months treatment may be needed), 12 monthly doses in 18 months are considered enough.
- Single lesion PB children 5-14 years: Rifampicin 300 mg, Ofloxacin 200 mg, Single lesion PB adults: Rifampicin 600 mg, Ofloxacin 400 mg, Minocyclin 100 mg
- Minocyclin 50 mg (not recommended under age 5).
- In younger children treatment regimens should be adjusted according to age and weight.
- Always check for reactions and complications, particularly haemolysis in Northern and Western Europeans.
- RR: prednisolon 0,5 mg/kg daily, tapering down slowly but remaining above 0,25 mg/kg/day for 3-6 months according to clinical signs and symptoms, then taper down to zero in 2 months.

- ENL: Mild ENL: acetylsalicylic acid 1000 mg 3 times daily (or less according to age) for 1-2 weeks. Severe ENL: prednisolone high dose eg 1-1.5 mg kg for two days, tapering off in two weeks. To be repeated if necessary.

Clinical pictures

Girl with TT leprosy, hypopigmented patch with loss of sensation

11 year old boy with BT leprosy

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Skin infections

FUNGAL

Tinea capitis

Epidemiology

Fungal infections of the scalp (tinea capitis) are endemic among schoolchildren in tropical Africa and they can cause significant public health problems. The prevalence of tinea capitis is higher among schoolchildren in rural schools and schools with a lower socioeconomic status.[1-5]

Etiology and pathogenesis

It is an infection of the hair shaft on the scalp, which may be caused by *Trichophyton* and *Microsporum* species. It is predominantly a disease of prepubertal children and the incidence of *Microsporum* species is higher in boys than in girls. The causative agent of tinea capitis varies with geography, socioeconomic status and time.[6]

Antropophilic infections like *Trichophyton tonsurans, violaceum*, *sudanense and Microsporum audouinii*, are most prominent in Africa.

Clinical findings

The clinical appearance can vary from scaling (diffuse scaling with discrete patches of hair loss), hair loss, black dots and sometimes pustules, nodules to massive purulent secretion (Kerion).

Late detection and lack of treatment can result in widespread infections and, in rare cases, permanent alopecia. Because the fungus has grown into the hair follicle, systemic treatment is necessary.[7;8]

Differential diagnosis

- Seborrheic dermatitis
- Atopic dermatitis
- Tinea amiantacea
- Psoriasis
- Alopecia areata
- Trichotillomania
- CDLE
- Pyodermia

Management

- Oral antifungal treatment is always indicated

*** Griseofulvin**

- The dose is based on body weight and is usually 20 mg/kg of body weight once a day for 6-8 weeks. Oral solution (Syrup, griseofulvin microcrystalline, 25 mg/ml) or tablets (griseofulvin 125 mg or 500mg).
- Dosing recommendations have not been established for children < 2 years of age.
- Children above 30 kg 500 mg daily for 6-8 weeks.
- For tinea capitis Griseofulvin is the treatment of choice.

***Terbinafin**

- In certain countries not approved for children below 2 years of age.
- The dose is based on body weight.
- Children below 20 kg 62.5 mg daily for 6 weeks. (Syrup, terbinafin, 25 mg/ml), 2.5 ml Syrup daily or tablet (terbinafin 250 mg), ¼ tablet daily.
- Children between 20 and 40 kg 125mg daily for 6 weeks. Oral solution (Syrup, terbinafin, 25 mg/ml) 5 ml daily or tablets (terbinafin 250 mg) ½ tablet daily.
- Children above 40kg and adults 250 mg daily for 6weeks. Tablets (terbinafin 250 mg), 1 tablet daily.
- To prevent shedding, apply Whitfield's cream or miconazole cream twice daily topically, preferably after shaving or use anti-fungal shampoo (like 2% ketoconazole or 2.5% selenium sulfide).

Clinical pictures

Scaling and hair loss

Secondary infection

Kerion and possible permanent alopecia

- Infected siblings and friends of affected children should also be treated.
- Appropriate adjunctive treatment for household contacts includes daily use of an antifungal shampoo.
- In case of a secondary bacterial infection oral antibiotics like cloxacilline or erythromycine can be given. For dosages see impetigo.

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Tinea corporis

Epidemiology

Superficial fungal infections ("ringworm") of the skin are common in sub-Sahara Africa, especially on exposed skin surfaces, though tinea corporis is less common than tinea capitis and pedis. It is found mostly in rural areas.[1;2]

Etiology and pathogenesis

Zoophilic fungal infections like *Microsporum canis* and *Trichophyton* verrucosum normally present on the exposed surfaces of the body like the face, arms and shoulders. On the trunk and the legs antropophilic infections like *Trichophyton tonsurans, violaceum, sudanense and Microsporum audouinii*, which are most prominent in Africa, are more frequently found.[3;4]

Clinical findings

Tinea corporis presents as typical round lesions with central healing, hair loss and scaling on the edges. They can be large and widespread, due to lack of treatment or in case of immunosuppression. The clinical and social impact of fungal infections on individuals varies with local conditions.[5-7]

Differential diagnosis

- Eczema
- Pityriasis versicolor / rosea
- Granuloma annulare
- Psoriasis
- Acne vulgaris
- Leprosy

Management

- Application of an imidazole cream or Whitfield's ointment twice daily for 6 weeks.
- In case of large and multiple lesions oral treatment with griseofulvin or terbinafin is preferred during 2-4 weeks. See for the dosages tinea capitis.

Clinical pictures

Widespread round lesions

Localized lesion, central healing and scaling on the edges

Reference List

1. Hogewoning A.A., et all. Skindiseases among schoolchildren in Ghana, Gabon and Rwanda. August 2012; Accepted for publication in the *International Journal of Dermatology.*
2. Komba EV, Mgonda YM. The spectrum of dermatological disorders among primary school children in Dar es Salaam. *BMC Public Health* 2010; **10**: 765.
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Tinea pedis (Interdigital type)

Epidemiology

Hot, humid climate and a changing, more western lifestyle of wearing closed shoes makes tinea pedis an increasing problem among African schoolchildren, especially in urban areas. The prevalence rate is still low.[1;2]

Etiology and pathogenesis

Trichophyton rubrum, mentagrophytes and *Epidermophyton floccosum* account for most cases of tinea pedis. In a tropical environment *Hendersonula toruloidea* is also frequently involved. Interdigital infections are often mixed infections of the above mentioned fungi and bacteria (*Nocardia minutissima*) which can cause erythrasma. Dermatophyte infection provides a portal of entry which may lead to bacterial infection with *Streptococci or S.aureus*.[3;4]

Clinical findings

Tinea pedis or athlete's foot causes cracking, maceration and inflammation with itching between the toes, most commonly between the 4th and 5th toe.[5]

Differential diagnosis

- Erythrasma
- Bacterial infection
- Eczema (dyshidrotic or contact allergic)

Management

- Topical treatment is always necessary.
- An imidazole containing cream, ciclopirox cream twice daily or terbinafine cream once daily for 6 weeks or longer, until a week after the symptoms subside. The web spaces between the toes should be kept dry, especially after washing. Also cotton socks should be used and changed daily.
- When the complaints are often recurring, the inderdigital spaces can be treated twice weekly with the above mentioned creams as prophylaxis.
- Oral antifungals alone are usually ineffective because topical treatment is essential and infections are often mixed.
- Children should be advised to wear well ventilated shoes.
- If there is a superimposed bacterial infection, topical antibiotic treatment can be applied like Gentian violet paint 0.5%, mupirocin ointment or betadine ointment twice daily on the lesions. In severe cases oral antibiotics can be given like cloxacillin or erythromycin. For dosages see impetigo.

Clinical picture

Typical white macerated lesions of Athlete's foot

Reference List

1. Hogewoning A.A., et all. Skindiseases among schoolchildren in Ghana,Gabon and Rwanda. August 2012. Accepted for publication in the *International Journal of Dermatology*
2. Komba EV, Mgonda YM. The spectrum of dermatological disorders among primary school children in Dar es Salaam. *BMC Public Health* 2010; **10**: 765.
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Pityriasis versicolor

Epidemiology

Pityriasis versicolor is a chronic benign fungal infection frequently seen among young adults, more commonly in a tropical environment.[1;2]

Etiology and pathogenesis

Pityriasis verscicolor is caused by the yeast *Malassezia* which is a normal resident of the skin and is usually asymptomatic.[3] In favorable circumstances such as a hot and humid climate, and / or sweating the infection becomes symptomatic.

Clinical findings

Clinically it is characterized by well-defined scaly hypo-or hyper pigmented patches primarily affecting the upper trunk, neck or upper arms, in areas with active sebaceous glands.[4] In longstanding disease the patches become confluent and may cover large

areas. After treatment hypopigmented macules without scaling may persist but these will disappear after sun exposure.

Differential diagnosis

- Vitiligo
- Pityriasis alba
- Sarcoidosis
- Epidermodysplasia verruciformis
- Verrucae planae (in a HIV+ patient)

Management

- Avoid the use of vaseline, olive oil and other greasy products.
- Ketoconazol, miconazol or terbinafin cream twice daily on the lesions for 3 weeks.
- Apply selenium sulphide shampoo as a lotion on the whole body overnight, wash off in the morning and wash the scalp extra.
- Selenium sulphide shampoo or ketoconazole 2% shampoo daily for 7 days or twice weekly for 4 weeks. The shampoo should be left on the skin for at least 15 minutes before being rinsed off.
- Salicylic acid 5% + sulphur 5% ointment during the night for 4 weeks.
- Recurrences can be prevented by once monthly preventive treatment with any of the above mentioned medications.
- Because of the risk of hepatotoxicity and the high recurrence rate in the tropics oral treatment should be avoided among children.

Clinical pictures

Hypopigmented macules

Hypopigmented patches with fine scaling

Hypopigmented patches with fine scaling

Reference List

1. Komba EV, Mgonda YM. The spectrum of dermatological disorders among primary school children in Dar es Salaam. BMC Public Health 2010; **10**: 765.
2. Mahe A, Hay R. Epidemiology and management of Common Skin Diseases in Children in Developing Countries. (http://whqlibdoc.who.int/hq/2005/WHO_FCH_CAH_05.12_eng.pdf). Dec 2005.
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Skin infections

VIRAL

Verrucae vulgares

Epidemiology

Common warts are caused by a small group of Human Papilloma Virus types. They penetrate the skin after skin to skin contact or through contaminated surfaces and objects (e.g. at home, public showers, swimming pools). Prevalences of 20% are reported among schoolchildren in industrialized countries although the prevalences found in most community based studies in sub-Sahara Africa are much lower.[1-5]

Etiology and pathogenesis

Papillomavirus infect squamous epithelia of the skin and mucous membranes in most vertebrate species. Many types of HPV have been identified and are associated with various clinical lesions. HPV types 1, 2 and 4 infect the skin and induce common warts. They are found at any age but are most common in teenagers. The extent of lesions is determined by the immune status of the host.[6]

Clinical findings

The lesions are discrete, round papules and nodules with verrucous surface. They can be small papules (1-10mm) or large plaques. Sometimes the lesions become confluent and form a mosaic. In the majority of patients with a normal immune system warts will disappear spontaneously within several months to years. Treatment is sought for when lesions are painful (eg on the soles) or unsightly but is not always necessary. Treatment results are unpredictable and often disappointing. Warts may spread fulminantly and persist indefinitely.[7;8]

Differential diagnosis

- Lichen planus
- Psoriasis
- Plantar callus (corns)
- Mollusca contagiosa
- Verrucous tuberculosis

Management

- Apply salicylic acid 25% ointment daily (possibly under occlusion) and cut the warts with a razorblade. Repeat this for weeks to months.
- Apply trichloro or monochloro acetic acid.
- Freeze with liquid nitrogen. Warn the patient for post-treatment hypo or de pigmentation which is usually temporary. (see picture 2).
- Curettage (after local analgesia with Emla cream).
- Laser only in specialized centers.

Clinical pictures

Hyperkeratotic papules and nodules

Depigmentation after cryotherapy

The differential diagnose with Lichen planus is not always easy. ...

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Mollusca contagiosa

Epidemiology

Mollusca contagiosa are frequently seen in children under the age of 5 years which can be the reason of a low prevalence found among schoolchildren in sub-Sahara Africa.[1-3] They can be localized anywhere on the body but are often seen in areas of warmth, moisture and friction such as the armpits and groins. In cooler climates the infection seems to be more common at a later age. The use of public swimming pools has been correlated with childhood infections.[4]

Etiology and pathogenesis

It is a common cutaneous infection caused by a pox virus and can affect both children and adults. The virus can be transmitted directly from person to person or by autoinoculation, the incubation time can vary from weeks to months. In adults it is regarded as a sexually transmitted infection and one should consider the possibility of co-existent HIV infection. Therapy is not always necessary but may be beneficial in preventing transmission or autoinoculation.[5-7]

Clinical findings

Pearl-like, dome shaped nodules with a dimple on top can be seen, the diameter varies from 5 to 10 mm. If squeezed a white/yellow greasy mass comes out of it. Sometimes a single lesion can be seen but normally there are several and sometimes hundreds. Most cases are self-limiting within 6-9 months.

Differential diagnosis

- Verruca vulgaris
- Milium
- Histiocytoma
- Keloid
- Adenoma sebaceum
- Cryptococcosis
- Tricholemmoma

Management

- Most treatment options are mechanical, sometimes causing discomfort but in the majority of cases therapy is not necessary and natural resolution can be awaited.
- Curettage with a sharp curette after applying 1% iodide tincture. Local anesthesia can be accomplished after application of Emla cream during 30 minutes.
- Cryotherapy with liquid nitrogen to be repeated every 3 weeks.
- Prick the center with a toothpick and press out the contents.
- Apply 50-88% trichloro acidic acid.
- Apply retinoid cream / tincture 0.05-0.1% 2 times daily.

Clinical picture

Little boy with "pearl" like nodules

Reference List

1. Hogewoning A.A., et all. Skindiseases among schoolchildren in Ghana,Gabon and Rwanda. August 2012: Accepted for publication in the *International Journal for Dermatology*
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Varicella / Chickenpox

Epidemiology

Varicella zoster virus (VZV) has a worldwide distribution, 98% of the adult population is seropositive. The first manifestation of a VZV infection is varicella (chickenpox). Varicella affects 90% of unvaccinated children under 10 years of age and less than 5 % over 15 years. Several point prevalence studies in Africa showed low percentages but epidemics occur seasonally.[1-3] It predisposes to the development of herpes zoster later in life. Immunization reduces the incidence of herpes zoster markedly.[4-6]

Etiology and pathogenesis

Varicella is very contagious and is spread by airborne droplets or direct contact with vesicular fluid. After primary infection it moves from cutaneous and mucosal lesions to dorsal root ganglion cells. From there it can be reactivated in a later stage.[7]

Clinical findings

Prodromes of primary varicella vary from mild fever to general malaise and are followed by multiple pruritic, erythematous papules and vesicles which become pustules and hemorrhagic crusts. From the scalp and face they spread to the trunk and extremities. Any numbers of vesicles varying from a few to several hundreds are seen in all stages of development at the same time. Itch is the major complaint and scratching may lead to secondary infection. The disease is normally self- limiting and lesions heal in 7 to 10 days. Common complications are secondary infection with scarring and pneumonia. In immunocompromised patients varicella can lead to severe morbidity and even death (see picture 3).[8]

Differential diagnosis

- Disseminated herpes simplex infection
- Disseminated herpes zoster infection
- Hand, foot and mouth disease
- Insect bites and scabies
- Bullous impetigo
- Pityriasis lichenoides et varioliformis acuta (PLEVA)

Management

- Calamine lotion or phenol-zinc lotion as necessary for itch and drying in.
- Sedating oral antihistamines like piriton and phenergan. For the dosages see urticaria.
- Limited secondary infection: use betadine scrub, apply betadine ointment, fucidin cream or ointment, mupirocin ointment, sulphur 5% in zinc oxide cream or gentian violet paint 0.5% 2 times daily.
- Severe or widespread bacterial secondary infections can be treated with systemic antibiotics like cloxacillin or erythromycin. For the dosages see impetigo.

- Immunization has 80% effectivity. Recommended for HIV+ children or children on HAART.[9]
- In immunocompetent children oral medication with aciclovir is only indicated in severe infections.
- Immunocompromised children should be referred to a specialist.

***Aciclovir**

- Child under 2 years: 200 mg 5 times daily for 5 days. Oral suspension (Syrup 40mg/mL or 80mg/mL) 5 ml (40mg/ml) 5 times daily for 5 days.
- Children between 2-5 years: 400mg 5 times daily for 5 days. Oral suspension (Syrup 40mg/mL or 80mg/mL) 5 ml (80mg/ml) 5 times daily for 5 days or tablets 400 mg 5 times daily for 5 days.
- Children above 6 years of age 800 mg 5 times daily for 5 days. Tablets 400mg 2 tablets 5 times daily for 5 days.

Clinical pictures

Widespread lesions on the back. The different stages of lesions, arising over 7 to 10 days, is typical of varicella.

Multiple, pruritic, papules, vesicles and pustules on the face of a young female. The different stages of lesions, arising over 7 to 10 days, is typical of varicella.

This HIV positive boy died two days later due to a generalized infection.

***Valaciclovir**: The dosage which is recommended in pediatric patients who are at least 2 years old to less than 18 years is 20 mg/kg administered 3 times daily for 5 days. The total dose should not exceed 1 gram 3 times daily.

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Herpes zoster

Epidemiology

Varicella Zoster Virus (VZV) can produce two different clinical manifestations, varicella (chickenpox) and herpes zoster (shingles). The point-prevalence of both skin diseases found among schoolchildren in sub-Sahara Africa was low,[1;2] most probably because affected children tend to stay at home. Chickenpox is primarily a disease of children and shingles a disease of adults but they may both occur at any age. VZV is distributed worldwide and 98 % of the adult population is seropositive. These figures become lower after vaccination campaigns.[3;4]

Each person with a history of varicella has approximately 20% chance of acquiring shingles in his/her lifetime. These figures are much higher in those infected with HIV.[5-7]

Etiology and pathogenesis

During the course of a primary varicella infection the VZV spreads from the skin and mucosal lesions into the sensory nerve endings. Reactivation of the VZV may occur spontaneously or may be triggered by fever, trauma, stress or immunosuppression. It can spontaneously lead to a clinical herpes zoster which is usually more severe in young children than in adults. Herpes zoster is more severe in the immune suppressed. [6;7]

Clinical findings

Herpes zoster can be preceded by a severe itchy, burning pain sensation in the involved dermatome. This prodrome may also consist of fever, headache and general malaise. The rash which develops within a sensory dermatome starts with erythematous macules and papules which later progresses to vesicles, pustules and crusts. There may be secondary bacterial infection. Especially in Africans the infection can lead to the formation of keloids but this is uncommon among immunocompetent children. Herpes zoster can be complicated by post herpetic neuralgia (rare among children). Herpes zoster of the ophthalmic branch of the facial nerve may be complicated by keratoconjunctivitis and lead to blindness, therefore ophthalmologic care should be sought.[5;6;8]

Differential diagnosis

- Insect bites
- Papular urticaria
- Herpes simplex (especially in the genital area)
- Contact dermatitis
- Localized bacterial or viral infections

Management

In a healthy child usually analgesics and local therapy are sufficient.

- Calamine or phenol-zinc lotion for vesicular stage.Gentian violet paint 0.5% may be used as well.
- Use betadine scrub/shampoo as a soap to prevent secondary infection. Do not use vaseline.
- In case of a secondary infection oral antibiotics like cloxacillin or erythromycin can be given. For the dosages see impetigo.
- Refer to the eye specialist when the eye is involved.
- In immunocompetent children oral medication is only indicated in severe infections.
- Immunocompromised children can better be referred for treatment with aciclovir or valaciclovir.

*Aciclovir

- Child under 2 years: 200 mg 5 times daily for 5 days. Oral suspension (Syrup 40mg/mL or 80mg/mL) 5 ml (40mg/ml) 5 times daily for 5 days.
- Children between 2-5 years: 400mg 5 times daily for 5 days. Oral suspension (Syrup 40mg/mL or 80mg/mL) 5 ml (80mg/ml) 5 times daily for 5 days or tablets 400 mg 5 times daily for 5 days.
- Children above 6 years of age 800 mg 5 times daily for 5 days. Tablets 400mg 2 tablets 5 times daily for 5 days.
- *Valaciclovir: The dosage which is recommended in pediatric patients who are at least 2 years old to less than 18 years is 20 mg/kg administered 3 times daily for 5 days. The total dose should not exceed 1 gram 3 times daily.

Clinical pictures

Multiple vesicles and open lesions on a leg of a 3 year old boy

Multiple vesicles following a dermatome

Herpes zoster of the shoulder

Reference List

1. Hogewoning A.A., et all. Skindiseases among schoolchildren in Ghana, Gabon and Rwanda. Aug.2012; accepted for publication in the *International Journal of Dermatology.*
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Herpes simplex

Epidemiology

Herpes simplex infections commonly take place in early childhood and are often asymptomatic. The clinical prevalence among schoolchildren is low. More than 60% of infected children remain carriers of the virus. Percentages of 90% antibodies against herpes simplex virus 1 (HSV1) have been found among young adults worldwide. The percentage of antibodies against HSV2 is much lower before adolescence as it is usually sexually transmitted. Minor epidemics of HSV1 may occur among schoolchildren or in nurseries.[1;2]

Etiology and pathogenesis

HSV1 is usually transmitted by direct contact with saliva. After an often asymptomatic infection viral elements persist as a latent infection for life. Reactivation occurs during periods of immunosuppression like fever, stress, exposure to UV light, HIV infection, menstrual period or infectious diseases like malaria.[3] Recurrent herpes simplex is common and may be the result of an endogenous reactivation or an exogenous reinfection.[1;4]

Clinical findings

Most infections are asymptomatic. Symptomatic primary infections in children often present as a gingivostomatitis together with an increased salivation and difficulty in eating. On the lips multiple painful, burning vesicles appear and develop into pustules and ulcerations. In recurrent infections the clinical picture is usually much milder.[1]
In HIV positive children the symptoms are more severe and the attack rates are higher but this improves after initiation of HAART.[5;6]
In cases of genital herpes in young children sexual abuse should be considered.

Differential diagnosis

- Angina
- Aphthous stomatitis
- Oral candidiasis
- Fixed drug eruption

Management [7]

In case of a primary infection analgesics are indicated.

- In recurrent infections: A lip cream / stick with a sun blocker daily to prevent recurrences.
- In immunocompetent persons oral medication with aciclovir is only indicated in severe primary infections or very frequent recurrences. For dosages see varicella/chicken pox.
- In the immunesuppressed oral (val)aciclovir is recommended.
- Warn for meningitis.

***Aciclovir** (British National Formulary)

- Child under 2 years: 200 mg 5 times daily for 5 days. Oral suspension (Syrup 40mg/mL or 80mg/mL) 5 ml (40mg/ml) or 2.5 ml (80mg/mL) 5 times daily for 5 days.
- Child over 2 years adult dose: 400mg 5 times daily for 5 days.
  Oral suspension (Syrup 40mg/mL or 80mg/mL) 10 ml (40mg/ml) or 5ml (80mg/mL) 5 times daily or tablets (200mg) 5 times daily for 5 days.

***Valaciclovir**: The dosage which is recommended in pediatric patients who are at least 2 years old to less than 18 years is 20 mg/kg administered 3 times daily for 5 days. The total dose should not exceed 1 gram 3 times daily.

Clinical pictures

Herpes simplex with secondary impetigenisation in a caucasian boy

Secundary infected Herpes simplex lesion in a HIV+ child

Herpes simplex infection of index finger in a small child

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Skin infections

PARASITIC

Cutaneous Leishmaniasis

Epidemiology

Leishmaniasis in its various forms is present on all continents except Australia and Antartica.[1] It is a widespread disease with 350 million people at risk. There are around 2 million new cases a year of which 500,000 cause visceral and 1,500000 cause (muco) cutaneous disease.[2;3] The epidemiology and age range affected depend on the characteristics of the parasite species and exposure history. In areas with high transmission rates the adult population will generally have acquired immunity and more children will be affected.[4] Most CL cases occur in the Middle East and in South and Central America but it is also endemic around the Mediterranean basin and in South Sudan, Kenya and especially Northern Ethiopia.[5;6]

Etiology and pathogenesis

The leishmaniases are a complex of diseases caused by the intracellular protozoaLeishmania. Disease transmission occurs through the bite of an infected sandfly. CL in Africa is mainly caused by *Leishmania major*. There are smaller foci of *L. tropica, L. infantum and L. Aethiopica*.

CL usually affects the exposed skin, of the face, neck and arms. Poorly functioning health care facilities, poverty and lack of knowledge all play a role in the spread of leishmaniasis.[3;4;6;7]

Clinical findings

One week to three months after an infected bite or bites solitary or multiple lesions appear. A red or skincoloured papule develops into a non-healing plaque or nodule which often shows central ulceration with a well demarcated with a violaceous border. It is usually painless unless superinfected. Regional lymphatic tissue can be involved, leading to a lymphadenitis. Untreated it usually leaves an ugly atrophic scar. Continuous ulceration and diffuse cutaneous infection may occur.

Differential diagnosis

- Leprosy
- Impetigo / ecthyma
- Insectbite
- Cutaneous tuberculosis
- Atypical mycobacterial infections
- Syphilis

Management [4]

- The choice of treatment depends on the type of leishmania and the number of lesions.
- Preventive measures like protective clothing and avoidance of bites.
- When there are single or limited number of localized lesions cryotherapy, electrocoagulation and surgery are treatment options.
- Single or limited number of lesions Pentostam or sodium stibogluconate (SSG): 6 to 10 times once or twice weekly intralesional injections (inject 0.5 to 1.5 ml of 100 mg/ml).
- Glucantine or Pentostam 20 mg/kg/day for 20-30 days i.v or i.m.
- Pentamidine 4mg/kg/weekly i.m. as long as necessary in cases of diffuse cutaneous leishmaniasis by *L. aethiopica*.
- Miltefosine, for children from 3 years and older: 1.5-2.5 mg/kg/daily orally for 28 days.
- Itra-, keto-, fluconazole depending on species.
- Amphotericine B (Amphotericin B and liposomal Amphotericin B are especially effective in visceral leishmaniasis and PKDL and should be administered in a specialized centre).

Clinical picture

Typical lesion on the cheek of a young east African boy

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Scabies

Epidemiology

Scabies is a common ectoparasitic infestation caused by *Sarcoptes scabiei,* a human-specific mite that is highly prevalent in some areas of the developing world, though the prevalence of infection in communities may be cyclical.[1-3]

Etiology and pathogenesis

It is mostly spread by close personal contact but can also be spread by clothing, sheets and towels. Secondary bacterial infection of scabies is common and might increase the risk for glomerulonephritis.[4;5]

Clinical findings

Typical sites of involvement are the interdigital spaces of the hands, the flexural parts of the wrists, the armpits, the feet and the genitals. One of the clinical signs, though not always present in warm climates, is the burrow (S- shaped ridge) caused by the excavation of the female mite for her eggs. Small erythematous papules can be present together with excoriations. Itching, especially at night, is the main complaint which often results in scratch marks and secondary infection.[6-8]

Differential diagnosis

- Eczema
- Contact dermatitis
- Pyoderma
- Bullous pemphigoid
- Insect bites
- Papular urticaria

Management

- Treat all individuals living in same household at the same time.
- Wash sheets and clothes or hang them outdoors for at least 24 hours.
- Sulphur ointment 5-10% to apply twice daily for at least one week.
- Benzyl benzoate emulsion (10 to 25%) is applied over the entire body and left on the skin for up to 24 hours before washing off. Treat during 3 nights and repeat after one week.
- Epidemics in institutions like prisons and boarding schools may be treated with Ivermectin on day 1 and day 10 . Not suitable for children below 5 years of age. See for the dosages cutaneous larva migrans.
- In case of secondary infection oral antibiotics like cloxacillin or erythromycin can be given. For the dosages see impetigo.
- For severe itchiness sedating oral antihistamines like Piriton or promethazine can be used. For the dosages see urticaria.
- After treatment complaints of itch may persist for weeks. This can be treated with mild topical steroids like hydrocortisone cream or ointment two times daily.

Clinical pictures

Interdigital papules

Papules, pustules and scratchmarks

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Skin infections

HELMINTH

Cutaneous larva migrans/creeping eruption

Epidemiology

Cutaneous larva migrans (CLM) is endemic in resource-poor communities in the developing world and occurs sporadically in high-income countries, where it is commonly seen as an imported skin disease in travelers[1-4]

Etiology and pathogenesis

Cutaneous larva migrans (CLM) in humans is usually caused by the penetration of cat or dog hookworm larvae into human skin. The presence of animal reservoirs like cats and dogs ensures ongoing transmission. Anyone walking barefoot or sitting on a contaminated beach is at risk. Transmission occurs when skin is in direct contact with soil, contaminated by dog or cat faeces and/or urine or indirect via towel and underware. Humans become a dead-end host because the migrating parasite cannot penetrate into the dermis and eventually dies in the epidermis. Its cutaneous manifestations usually resolve within weeks or months. [2;5]

Clinical findings

The lesions are characteristically urticarial, raised and vesicular. The diagnosis is made clinically in the presence of a linear serpiginous track moving forward in the skin at a speed of 1 to 5 cm per day. The lesions can be intensely pruritic and bacterial super infection often occurs as a result of scratching. Most lesions are located on the trunk, legs, and feet.[6;7]

Differential diagnosis

- Larva currens (Strongyloides stercoralis infection)
- Folliculitis
- Scabies and other ectoparasites
- Insect bites
- Urticaria

Management

- Cryotherapy with liquid nitrogen may be tried for limited lesions. Treat the skin at 1 cm ahead of the visible trail; this is where the larva is found.
- For oral treatment the use of ivermectin or albendazole can be considered.

Ivermectin

- Preferably not for children below 5 years of age.
- Dosage depends on bodyweight and is usually given in a single dose.
- Child between 15-25 kg: 1 tablet of 3 mg ; Child between 25-35 kg: 2 tablets of 3 mg; Child between 35-50 kg: 3 tablets of 3 mg; Child between 50-65 kg 4 tablets of 3 mg; Child above 65 kg: adult dose 5 tablets of 3 mg

***Albendazole**

- Children below 2 years of age: 200 mg once or twice daily for 1 to 3 days. Oral suspension (40 mg/mL) 5 ml syrup once or twice daily for 1 to 3 days. Children of 2 years and above: 400 mg once or twice daily for 1 to 3 days. Oral suspension (40mg/mL), 10 ml syrup once or twice daily for 1 to 3 days or tablets (200mg), 2 tablets once or twice daily for 1 to 3 days.
  - Secundary infection can be treated with betadine scrub, potassium permanganate solution or Gentian violet paint.

Clinical pictures

Cutaneous larva migrans on the leg of a Kenian toddler

Cutaneous larva migrans detail

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Lymphatic Filariasis

Epidemiology

Lymphoedema in the tropics can have several causes, but is usually caused by inflammation and consequently adenitis due to bacteria, fungi or minerals. Secondary lymphoedema due to filariasis has a high prevalence and is considered by many the most prevalent cause. In Africa *Wuchereria bancrofti,* a parasitic worm infection transmitted by mosquitoes, is responsible for the majority of cases of lymphatic filariasis. Out of the 120 million infected patients worldwide 40 million develop clinical symptoms.[1-3]

Etiology and pathogenesis

Lymphatic filariasis is a helminth disease that causes chronic and long-term infection with host inflammation due to the antigenic determinants of the parasite. Adult worms are present in the lymphatics and the resulting inflammatory response can cause obstruction. This obstruction is often acquired in childhood and leads to acute attacks of dermato-lymphangio-adenitis and elephantiasis, lymphoedema of limbs and genitals. Like in other filarial infections the symbiosis with the Wolbachia bacteria is likely to be essential for the multiplication and development of the parasite.[2;4]

Clinical findings

After an incubation period of 5 to 15 months the presence of adult worms can lead to lymphangitis and lymphadenitis with localized pain and pitting oedema starting in the upper legs. These attacks can become chronic and cause lymphatic vessel dysfunction and damage.[2;4-6]

Differential diagnosis

- Kaposi sarcoma
- Lymphoedema caused by bacterial or fungal lymphangitis.
- Lymphoedema caused by podoconiosis (silicates in red volcanic soil which enter the soles and block the lymphnodes after an inflammatory reaction).[6]

Management

- Prevent secondary infections.
- Lymph massage, elastic compression bandages and or stockings.
- (Breathing) exercise. [7]
- Ivermectin plus albendazol in a single dose. To be repeated yearly during 5 years.[2;8;9] For the dosages see cutaneous larva migrans.
- Together with the ivermectin and albendazol treatment, a 6-week course of doxycycline (100–200 mg per day) has been recommended (not in young children). This treatment serves to reduce the Wolbachia bacteria but is still under discussion.[2;8;10]

Clinical picture

A late consequence of filariasis

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Onchocerciasis

Epidemiology

Onchocerciasis is a chronic tropical parasitic disease, caused by the nematode *Onchocerca volvulus*, most widely known for causing "river blindness" and severe dermatological problems.[1] It is found in 28 African countries with the highest prevalences in sub-Saharan West African nations like Ghana, Nigeria, Liberia and Mali.[2] Around 17 million people are affected worldwide.[3] In Africa, where the the burden of onchocerciasis is greatest, years of treatment and eradication programmes have led to a dramatic decrease of transmission.[4;5]

Etiology and pathogenesis

The vector of *Onchocerca volvulus* is the *Simulium* or black fly which lives close to fast moving, oxygen rich water. After infection it takes 12 to 18 months before the first

clinical signs present. The female larvae develop to adulthood and form fibrous capsules, the so called onchocercomata. During adulthood, the female worm sheds hundreds of thousands of microfilaria which migrate through the skin of the human host and cause severe itch, and, after repeated infections, in some regions blindness. Like in other filarial infections symbiosis with the Wolbachia bacteria is essential for multiplication and development of the parasite. A biopsy or skin snip test may show microfilaria.

Clinical findings

The most common skin problem in the first stage is troublesome itching with some erythematous hyper pigmented papules and patchy lichenification. In the chronic stage there can be pruritic generalized lichenification and depigmentation ("Leopard skin") later on. Sub dermal nodules ("onchocercomata,") are mostly seen over bony prominences like the hips but can be present anywhere. The loss of elasticity may cause so-calles hanging groins and lymph edema.[7;8]

Differential diagnosis

- Food allergy
- Other parasitic infestations
- Leprosy
- Syphilis

Management

- The standard treatment is ivermectin orally every 6 to 12 months). For the dosages see cutaneous larva migrans. Single-dose ivermectin effectively kills microfilariae but has little effect on adult worms; therefore, it controls but does not cure the disease.
- A patient staying in an endemic area needs treatment every 3 to 12 months, not only to kill new microfilaria but also for the treatment of reinfection.

Clinical pictures

Itch and lichenification

"Leopard" skin

- Together with the ivermectin treatment a 6-week course of doxycycline (100–200 mg per day) given to eliminate the Wolbachia bacteria. Because of the deposition of tetracyclines in growing bone and teeth it should not be given to children under 12 years or to pregnant or breast-feeding women. [2]

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Inflammatory skin diseases

Eczema/Atopic Dermatitis

Epidemiology

Eczema is widespread in the industrialized world and a growing clinical problem in sub-Sahara Africa.[1;2] In West Africa, the prevalence of eczema was considered to be < 5% although recent studies in West Africa and other parts of Africa have shown an increase, particularly amongst infants.[3;4] However, recent point-prevalence rates among schoolchildren in West and Central Africa, derived after physical examination by a dermatologist, were considerably lower.[5]

Etiology and pathogenesis

Atopic dermatitis or eczema is a chronic relapsing, pruritic inflammatory skin disorder. Although termed atopic, up to 60% of the children with the clinical phenotype do not

have demonstrable IgE-mediated sensitivity to allergens. Therefore it is preferable to use the term 'eczema'.[6;7]

Eczema is a multifactorial skin disease. Some risk factors for eczema are; genetic predisposition (like asthma and hay fever which may run in the family), emotional stress and change in lifestyle (such as changes in food patterns, contact with irritants or frequent washing).[8-11] Mutations in the filaggrin gene (FLG) are a major predisposing factor for ichthyosis vulgaris and eczema in individuals of European and Asian descent. These genetic findings provide an important support for the well known impairment of the epidermal barrier observed in eczema and could also deliver further clues to the natural history of the disease. Recent research indicates that FLG loss-of-function variants are less common in Africa.[12]

Clinical findings

Clinically eczema in the acute stage is characterized by itching, redness, oozing, crusts and often secondary infection with *Staphylococcus aureus*. The chronic stage is characterized by lichenification, excoriations and a very dry skin. Especially elbow-and knee folds, wrists, ankles, face and neck are affected.[13;14]

Three distinct clinical phases of eczema can be observed according to the age. In the infantile phase the eruption characteristically starts on the cheeks and scalp but the whole body can be affected. In the childhood phase especially the flexural areas of the knee and elbows are affected but also the wrists, ankles and buttocks can be involved. In the adult phase especially the neck and face are affected with a more diffuse scaling and erythema.Xerosis and lichenification are important characteristics.

Differential diagnosis

- Seborrheic dermatitis
- Contact dermatitis
- Psoriasis
- Scabies
- Dermatophytosis
- HIV related dermatoses

Management

- Explain the multifactorial and chronic character of the disease to the patients, parents and / or care takers.
- The use of soap and the frequency of washing should be reduced. Cotton clothing is preferred to wool or synthetics. Children should not be dressed too warm.
- Moisturize the skin regularly with an emollient cream or ointment like aqueous cream, coco butter or shea butter.
- In severe cases a potent steroid ointment like betamethasone can be applied once daily on the lesions.[15] Potent steroids should be used during a limited time and intermittently because of the risk of atrophy and bleaching. The use of potent corticosteroids should be avoided for use on the face or intertriginous sites like the groin or armpits.
- If available topical calcineurin inhibitors (TCI) like tacrolimus (0.03% and 0.1% ointment) or pimecrolimus (1% cream) can be used as maintenance treatment. The TCI don't cause skin atrophy. They may however cause a burning sensation upon application especially in the beginning of the treatment.
- In case of secondary infection oral antibiotics like cloxacillin or erythromycin can be given. For the dosages see impetigo.
- For severe itchiness sedating oral antihistamines like Piriton or promethazine can be used. For the dosages see urticaria.
- In more severe cases, when phototherapy or systemic therapy might be needed, patients should be sent to a referral / university hospital.

Clinical pictures

Eczema: elbow and knee folds, typical localizations

Eczema: detail: lichenification, hyper-pigmentation and scratch marks

Eczema: secondary infection

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Acne vulgaris

Epidemiology

Acne vulgaris is common in children and adolescents from age 10 and commonly persists up to age 25. In industrialized countries it affects between 30% and 100% of the adolescent population.[1-3] The prevalence of acne is considerably lower in developing countries though Westernization in urban areas in developing countries has been shown to lead to higher prevalence.[4-7]

Etiology and pathogenesis

Several factors play an important role in the etiology of acne: follicular epidermal hyper proliferation, excess of sebum production, activity of *Propionibacterium acnes* and inflammation. Androgenic hormones stimulate the hyperproliferation of the follicular keratinocytes and lead to an increased sebum production which is followed by the formation of comedones. Especially *P. acnes* is an important factor in the process of inflammation. A genetic predisposition, especially with the nodulocystic form has been

suggested. Recently some studies described a relationship between the development of acne and Body Mass Index.[4;8;9] The use of oil, bleaching and cosmetic creams is another important factor.

Clinical findings

The sites most affected are the face, back, chest and shoulders. Non inflammatory acne may consist of open comedos (blackheads) or closed comedos (whiteheads). In inflammatory acne the comedos expand to form erythematous papules, pustules, nodules or cysts. Pomade acne is very frequently seen in Africa due to the use of petrolatum or petroleum jelly (e.g. Vaseline as brand name). The nodulocystic form of acne can lead to severe scarring.

Differential diagnosis

- Folliculitis due to yeasts (Pityrosporum) or bacteria
- Perioral dermatitis
- Milia

Management

- Stop the use of oily cosmetics or petrolatum on the skin and hair.
- Apply benzoyl peroxide 5-10% preparations at night (because of its photosensitive effect) and warn the patient that it can bleach the pillows and pyjamas. Benzoyl peroxide preparations are available in creams, gels, lotions and washes.
- Apply topical retinoids at night (because of their photosensitive effect).Options are tretinoin (0.05% -0.1% solution or 0.02%-0.05% cream), adapalene and tazarotene (0.1% gel). Start at low concentrations to prevent irritation and hyperpigmentation.[10] Greater tolerability can be achieved by applying it the first two weeks of treatment on alternate nights.
- Apply topical clindamycin 1% lotion or erythromycin 2% lotion or gel in the morning.
- In case of moderate/ severe acne, use oral tetracyclines like tetracycline 250 mg twice or four times daily, doxycycline 100 mg once daily or erythromycin 4 times daily 250 mg and after one month 2 times daily 250 mg. The treatment has to be continued for several months and repeated when the acne comes back. Oral tetracyclines should not be given to young children.
- In case of moderate acne in women oral contraceptives may be given like Diane- 35 (cyproteronacetaat).
- Postinflammatory hyperpigmentation commonly occurs in a dark skin. Acne treatment should be started in an early phase in order to prevent this occurring.
- In case of severe acne or nodulocystic acne oral isotretinoin may be considered. The patient should be referred to a dermatologist for this treatment because of its potentially severe side effects among which teratogenicity.

Clinical pictures

A greasy skin and multiple papulopustules and comedones and postinflammatory hyperpigmentation

Pomade acne

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Psoriasis

Epidemiology

Psoriasis is a common skin disease in children although the prevalence is much lower than in adults. The total rate of psoriasis in children younger than 18 years found in Germany was 0.71% while this was 1.4% in Great Britain.[1;2] In the Netherlands and the US even lower figures were found and juvenile psoriasis seemed to be less common in the US among African Americans than among Hispanics and Caucasians.[3-5] The prevalence among girls is normally higher than in boys. Hospital based studies from Africa show prevalences of 1.5% in Egypt, 0.9% in Nigeria, 0.05% in Mali and 3.5% in Kenya.[6-9] Population based studies among schoolchildren in West and Eastern Africa showed very low prevalences.[10]

Etiology and pathogenesis

Psoriasis is characterized by the proliferation of keratinocytes and inflammatory cell infiltration of the dermis and epidermis. This reaction is caused by dermal infiltration of T lymphocytes and macrophages and leads to a fast turnover and hyperplasia of the epidermis. This results in a chronic inflammatory condition affecting the skin, nails, and joints.[11] Patients are genetically predisposed to psoriasis. Psoriasis in adults is associated with comorbidities such as obesity, hyperlipidemia, diabetes mellitus (metabolic syndrome), rheumatoid arthritis and Crohn's disease.[12] Physical trauma may trigger psoriatic lesions at sites of injury (Koebner's phenomenon).[4;11] Other triggers are antimalarials, lithium, beta blockers, stress, infections such as streptococcal angina and a cooler climate.

Clinical findings

The plaque type; this is the most frequently observed variant of psoriasis. It is characterized by sharply demarcated erythematous plaques covered by silvery white scales which shows the typical candle wax phenomenon after scratching. Lesions commonly appear on the elbows, knees, scalp, umbilicus, and lumbar area. The scalp is the most frequently affected site of involvement in pediatric psoriasis. Facial and intertriginous lesions may be difficult to differentiate from seborrheic eczema if there are no other typical psoriasis lesions.

Guttate psoriasis; is more frequently seen in children and consists of numerous papules and plaques (like "drops") all over the body. Guttate psoriasis is often preceded by a streptococcal throat infection.[11;13] The prognosis is good, with spontaneous remissions in weeks to months.

The inverse type of psoriasis; in this type of psoriasis the lesions appear as sharply defined erythematous plaques which show no or minimal scaling in intertriginous areas like the groin and armpits.

Erythrodermic psoriasis; nearly the whole body surface can be involved but this is rare in children.

Nail involvement (especially the fingernails) is uncommon in children with psoriasis. If it occurs nail-pitting is the common manifestation. Onycholysis and the "oil drop" sign are rare.[13]

Psoriatic arthritis; is an extracutaneous manifestation which is rare among children in Africa. A recent African review suggested an association between psoriatic arthritis and HIV infection.[9]

Differential diagnosis

- Tinea capitis and corporis
- Seborrheic dermatitis
- Eczema
- Lichen planus
- Pityriasis rosea / secondary syphilis (d.d. psoriasis guttata)

Management

- Discuss the chronic character ("come and go") of the disease with the patients and the parents / caretakers. Explain that psoriasis is not contagious but can be triggered by an infection. Natural sunlight has a beneficial effect.
- Approximately 70 to 80 percent of all patients with psoriasis can be treated adequately with use of topical therapy!
- Salicylic acid 5-10% in an oil, lotion, cream or ointment base 2 times daily to reduce the scaling.
- A moderate to strong topical steroid like betamethason ointment can be applied daily on the lesions. Cannot be used continuously for a long time because of side effects like atrophy and bleaching. Can be used in combination with salicylic acid 2-10% ointment.
- Coal tar 5-10% ointment or sulphur 5% in coal tar 5-10% at night.
- Vitamin D3 analogue like calcipotriol 2 times daily on the lesions, especially with plaque psoriasis. Can be used in combination with corticosteroids.
- Anthralin 0.1-1% cream or ointment. Especially for plaque psoriasis. Has to be wiped or washed off after 10-60 minutes. Not always suitable for children because of the irritative reaction.
- Find the possible bacterial sources of streptococcal infection (pharyngeal and perianal) and treat with antibiotics like erythromycin, penicillin or cephalosporines. For dosages see impetigo and ecthyma.
- If possible refer the patient to a dermatologist for phototherapy in the case of guttate psoriasis (UVB is the preferred form of phototherapy for pre-adolescent pediatric psoriasis).
- Systemic therapy with *Methotrexate, Ciclosporin, Retinoids* and *Biologicals* may be used for severe cases of chronic plaque psoriasis, guttate psoriasis in children who are unresponsive to antibiotics, topical treatment and UV therapy and to children with severe arthropathic psoriasis. These cases are rare and need to be referred to an university hospital or specialized centre.

Clinical pictures

Multiple hyperkeratotic plaques on the trunk and arms

Multiple hyperkeratotic, well circumscribed plaques on both knees

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Seborrheic dermatitis

Epidemiology

Seborrheic dermatitis is one of the most frequent skin disorders, especially the infantile form which affects as many as 70% of the newborns but disappears by the age of 1 year.[1;2] The prevalence in immunocompetent adults is between 1% and 3%, and is more common in men than in women. The prevalence is low in children over one and under 12 years of age[3;4] However in children of all ages, as in adults, it is frequently seen in combination with a HIV infection.[5]

Etiology and pathogenesis

The cause of seborrheic dermatitis is not completely understood. It occurs most often during periods of active sebum production (e.g., the neonatal period) and in areas of the skin where sebum is produced. There is no clear genetic predisposition but climate, stress and immunologic factors play an important role.[2;6]

Malassezia yeasts may play a role in the pathogenesis of seborrheic dermatitis since they are present on affected skin, and antifungal agents are useful in the treatment.[7] Especially in HIV-infection they appear to play a role.

Clinical findings

Seborrheic dermatitis is characterized by scaling and poorly demarcated erythematous patches that vary from pink yellow to red brown in color. In the African skin they are often hypopigmented. There is a predilection for places which are rich in sebaceous glands like the scalp, the nasolabial folds, glabella and the hairline, the sternum, the armpits and the groins.[2;6;8] The morphologic characteristics depend on the area of the skin involved. In healthy people the face and scalp are commonly affected, in the HIV-infected, armpits and groins also show lesions and they easily become superinfected. The lesions cause normally mild itching. Seborrheic dermatitis can give reason to social problems, especially with severe / moderate scaling of the scalp.[9;10]

Differential diagnosis

- Psoriasis
- Atopic dermatitis
- Tinea capitis

Management [2;11]

- In infantile seborrheic dermatitis the application of an emollient cream can be useful.
- Sulphur 3-5% cream to apply 2 times daily.
- Ketoconazole 2% cream or cicloporox 0.77% cream to apply 2 times daily.
- Ketoconazole or cicloporox shampoo. Low potency topical corticosteroids like hydrocortisone 1% cream 2 times daily, used intermittently.

- Topical calcineurin inhibitors like 0.1% tacrolimus ointment may be useful for facial lesions; it is too greasy for the armpits and groins.
- In severe and widespread lesions oral ketoconazole can be used. Usually it is given only to children above 2 years of age. The dosage in a child is 3 mg/kg daily for 2 weeks from 15 kg body weight onwards.

Clinical pictures

Bov 7 years old. HIV+ Seborrheic dermatitis of the scalp and the groin

Girl 6 years old, the differential diagnose with psoriasis capitis can be difficult.

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Lichen planus

Epidemiology

Lichen planus is frequently encountered in hospital based studies in sub-Sahara Africa.[1;2] In childhood it is unusual and pediatric patients comprise only 2% to 3% of all those affected.[3;4] There is no consistent gender predilection for childhood. In the USA it has been reported to be more prevalent among African American children.[5]

Etiology and pathogenesis

Lichen planus is an inflammatory dermatosis of unknown origin. Several reports have shown an association between lichen planus and liver disease such as chronic active hepatitis and as a complication of hepatitis B vaccination.[6] Also a positive history of auto immune diseases like myasthenia gravis, alopecia areata and lupus erythematosus has been described. In several studies there was a positive correlation with atopic dermatitis.[5] Quinine has also been described as initiating or worsening lichen planus.

Clinical findings

Lichen planus often presents with pruritic violaceous, polygonal, flat-topped papules and plaques most frequently seen on the flexor surfaces of the wrists and forearms (see picture 1) but the anterior side of the lower legs, the lumbo sacral region (see picture 2) and the neck are also common sites. Papules can develop at sites of trauma which represents the Koebner phenomenon. In the African skin, the lesions have a more grey aspect. With a drop of oil the striae of Wickham become visible. It can also affect the skin of the genitals and mucous membranes although this is very uncommon in young people. Lichen planus may resolve spontaneously with time ranging from a few months to years and often leaves residual areas of hyper pigmentation.[4;7]

Differential diagnosis

- Lupus erythematosus
- Lichen sclerosus and striatus
- Pityriasis rosea
- Secondary syphilis

Management [5;7;8]

- Parents and patients should be reassured that lichen planus is a benign non- infectious, self limiting disease.
- Moderate to strong topical corticosteroids like betamethasone 2 times daily, preferably combined with salicylic acid 5 % are the treatment of choice.
- Topical calcineurin inhibitors like tacrolimus 0.1% 2 times daily.
- In severe cases oral corticosteroids can be given (0.5-1 mg/kg daily) as a tapering dose over a 2-6 week period. Long term maintenance therapy with systemic corticosteroids should be avoided.
- Dapson 1mg/kg daily has been reported to be very helpful in severe cases.[9]
- Severe cases, unresponsive to treatment, should be referred to a dermatologist for phototherapy, intralesional therapy with triamcinolone 5-10 mg/ml, or systemic therapy with methotrexate or cyclosporine.

Clinical pictures

Multiple polygonal flat-topped papules and plaques especially on the wrists (differential diagnose verrucae vulgares!) and the lumbo sacral region.

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Alopecia areata

Epidemiology

Alopecia areata generally concerns pupils or students although the prevalence among schoolchildren in Africa was less than 1%.[1-4] The life-time risk of alopecia areata in the general population is approximately 1.7%. and in as many as 60% of patients the disease starts before the age of 20 years.[5] In patients with alopecia areata a considerable amount had episodes before or has a positive family history.[4]

Etiology and pathogenesis

Alopecia areata is an autoimmune disease that presents with nonscarring hairloss.[4] The pathogenesis is not completely clear.[6;7] Atopy, autoimmune thyroid disease, a positive family history and vitiligo are commonly associated. The course of the disease is unpredictable. Early and severe cases which last long have a less favorable prognosis.[8]

Clinical findings

Alopecia areata most commonly manifests as sudden loss of hair in a well demarcated, localized area in the scalp. The hair loss is usually limited to a single patch. The lesion is usually round or oval. “Exclamation point hairs” are frequently seen at the periphery of the lesion.[5]

The majority of patients present with limited alopecia. Approximately 80% present with one patch, about 12% with multiple patches on the scalp and possibly also in the eyebrows, lashes, and beard area, and about 7 % develop total baldness of the scalp (alopecia totalis), some even of all body hair (alopecia universalis).[9] The clinical diagnosis is made by the aspect of hairless patches with a normal skin.

Differential diagnosis [3;8;10]

- Androgenetic alopecia
- Traction alopecia
- Tinea capitis
- Trichotillomania
- Syphilis
- Atopic dermatitis
- Vitiligo

Management [5;11]

- Because of the high rate of spontaneous recovery a "watch-and-see" approach is often recommended.
- Psychological support may be offered if necessary.
- For patients who actively desire treatment, topical or intralesional corticosteroids are the treatments of choice. Betamethasone dipropionate lotion 0.05% can be applied 2 times daily for 12 weeks or betamethason cream 2 times daily for 1-2 months. If there is no improvement after 12 weeks the treatment should be stopped. Intralesional corticosteroids are appropriate for older children. Triamcinolon acetonide 10 mg/ml diluted with 2% lidocaine with epinephrine (to reduce the pain with the injections) can be injected intradermal once monthly and not longer than 6 months.
- Topical sensitizers like Anthralin (Dithranol) can be used in concentrations of 0.25-1% cream. Anthralin cream may be applied overnight, initially for 30 minutes and gradually to 1 hour. If there is no result it can be stopped after 3 months. Another possibility is the treatment with diphenylcyclopropenone (DPCP) but this is usually done under the supervision of a dermatologist.

Clinical pictures

Alopecia areata in a young child. A round well circumscribed area with hairloss...

Traction alopecia in a young girl...

- Ultraviolet A phototherapy (PUVA) is another option for which the patient has to be referred to a dermatologist.

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Pityriasis rosea

Epidemiology

Pityriasis rosea is a common, acute, self-limiting papulosquamous eruption. It typically affects children and young adults. There is a worldwide distribution and no ethnic predilection has been found. The prevalence among females seems to be slightly higher than males.[1] The point prevalence found among schoolchildren in Africa in several studies was low but in other, hospital based studies the period prevalences were higher.[1-3]

Etiology and pathogenesis

Pityriasis rosea is possibly of viral etiology ("flu of the skin"), it has been linked to human herpes virus 6 (HHV6). About a quarter of the patients have a history of a viral infection with upper respiratory symptoms shortly before or during the occurrence of the rash. Several medications can cause a rash similar to pityriasis rosea. It is self-limited and normally the eruptions last for 6 to 8 weeks.[4-6]

Clinical findings

Pityriasis rosea is characterized by an initial "herald patch" ("plaque mère"), followed by the development of a diffuse papulosquamous rash on trunk and arms. It can be difficult to identify until the appearance of characteristic smaller oval shaped secondary lesions that follow the cleavage lines. These lesions can form a so called "Christmas tree pattern" on the back. Pityriasis rosea is usually asymptomatic but can sometimes itch.[7;8]

Differential diagnosis

- Secondary syphilis
- Eczema (especially the herald patch)
- Psoriasis
- Tinea corporis

Management

- To differentiate between pityriasis rosea and secondary syphilis serologic testing for syphilis (VDRL or FTA-ABS) is necessary.
- Because in most cases it is self-limited and asymptomatic, a good explanation and reassurance of the patient is very important.
- In case of pruritus Calamine lotion or low to medium potent topical corticosteroids like hydrocortison1% or triamcinolon acetonide 0.1% cream can be applied 2 times daily.
- For severe itchiness oral sedating antihistamines like piriton or promethazine can be used. For the dosages see urticaria.
- Natural sunlight exposure can be beneficial.

Clinical pictures

Typical Christmas tree pattern on the back

More papulous pattern

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Benign skin tumors and nevi

Infantile Hemangioma (IH)

Epidemiology

Infantile hemangiomas are common, benign tumors of blood vessels, observed in 1-4 % of infants during the first year of life. Although most cases progress without problems, a small proportion can experience life-threatening complications.[1] They are more prevalent in female, caucasian infants and related with prematurity, advanced maternal age and multiple gestations.[2]

Etiology and pathogenesis

IHs are primarily composed of endothelial cells and can grow rapidly in the first 6 months of life, the proliferation phase. This phase can cause great concern to the parents. Normally it is followed by slow involution, leading to complete regression in about 70% of the patients in 5 to 10 years. The etiology of both stages is still not completely understood.[3;4]

Clinical findings

Most infantile hemangiomas occur within the first weeks of life. They vary in size from less than 1 cm to more than 10 cm. They can occur anywhere on the skin and mucosal surfaces though the preferred site is the face. Hemangiomas which are located in the superficial dermis are bright red in color ("strawberry ").Deep hemangiomas can be located in the deep dermis or subcutis and present as blue purple tumors. They may pose a problem during the growth phase when they can cause obstruction of vision or of the larynx or mouth.[5]

During the regression phase they can bleed easily or become necrotic. Ulceration is the most common complication occurring in approximately 15% of the patients. Regression can be complete and leaves no residual change at the site in most lesions (80%).In some areas it can leave atrophy, depigmentation, teleangiectasis and scarring.[6]

Differential diagnosis

- Capillary malformations or teleangiectasias
- Pyogenic granuloma
- Vascular malformations

Management

- Most cases need no treatment and have an excellent functional and cosmetic prognosis. Active intervention has to be avoided. Explanation to the parents / caretakers is essential.
- Proper follow up and management of ulceration. Local wound care with topical antibiotics like mupirocin or bacitracin ointments and occlusive dressings.
- When vital organs and functions like vision, hearing and breathing are impaired, the patient should be referred to a dermatologist and/or pediatrician for treatment with intralesional or systemic corticosteroids or interferon. Recent publications show good results with the treatment with local or systemic propanolol and systemic atenolol.[7;8]

Clinical pictures

A hemanioma on the lower lip in a Zimbabwean infant...

No problems feeding...

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Miscellaneous skin diseases

Oculocutaneous albinism (OCA)

Epidemiology

The prevalence of OCA is relatively low at general schools in sub-Sahara Africa.[1;2] Children affected with OCA are more commonly found in schools for the blind. Several prevalence studies in South Africa, Tanzania, Nigeria and Zimbabwe show figures of 1/5000-1/15000 but prevalences as high as 1 in 1000 were reported for selected populations.[1-6] The medical and social issues facing children with OCA are enormous and life expectancy is decreased compared with the general population.[7]

Etiology and pathogenesis

Oculocutaneous albinism (OCA) is an inherited functional disorder of melanin production which results in hypo or depigmentation of the skin, hair and eyes and extreme sensitivity to UV-damage. There are different types of OCA all of which have an autosomal recessive inheritance pattern.[7]

Clinical findings

People with OCA have a hypopigmented retina and fovea which leads to photophobia, nystagmus and lower vision. Exposure of the yellowish or white skin to the sun leads to sunburn, blisters, freckling and the formation of solar keratoses. Without sun protection measures basal and squamous cell carcinomas appear from the second or third decade[6;8;9]

Differential diagnosis

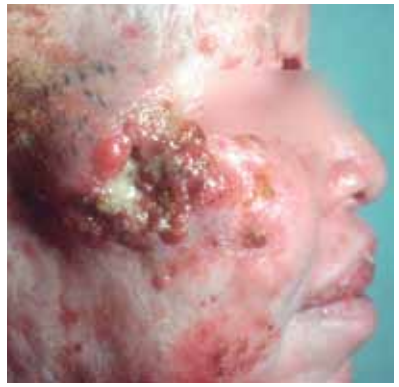
- Vitiligo
- Nutritional deficiencies

Management

- Protect the skin and eyes from sun damage: avoid the midday sun, wear a wide rimmed hat, protective clothing (long sleeves, long skirts and trousers) and sunglasses.
- Always use a sun block or a sun screen with a high sun protection factor (SPF) (PABA, zinc oxide, titanium dioxide).
- Use a sun block (e.g. zinc oxide or titanium dioxide) for the lips.
- Regular ophthalmological and dermatological check-ups.
- Treat solar keratoses with liquid nitrogen, curettage or topical 5% 5-fluoro-uracil.
- Malignancies should be excised, preferably in a specialized clinic.

Clinical pictures

Multiple lentigenes due to ultra violet damage



Without sun protection basal and squamous cell carcinomas appear in an early age...

...a vulnerable group of children

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Vitiligo

Epidemiology

Vitiligo may appear at any age. It affects around 0.5% of the world population. In hospital based studies from West Africa percentages between 2.8 and 6% have been presented.[1,2] In several community based studies among schoolchildren in Africa the prevalences were rather low.[3-6] The average age of onset found in a Nigerian study [7] was approximately 20 years.

Etiology and pathogenesis

The etiology of vitiligo is not exactly known though several studies point towards an autoimmune base, indicating the importance of a positive family history and the presence of other autoimmune diseases, such as diabetes mellitus and hyper-thyroidism.[7] There is an absence of melanocytes in the affected skin. Vitiligo is usually slowly progressive and seldom regresses spontaneously; sometimes the involved skin is pruritic.

Clinical findings

Vitiligo is characterized by sharply demarcated white macules surrounded by normal skin. It can be present on any part of the body but it is frequently localized on the face, the dorsal side of the fingers, the anogenital region and on sites of stretch and pressure. In an affected person it also occurs in traumatized skin, the Koebner phenomenon.
On darkly pigmented skin it is more obvious than on light skin. It can lead to a high level of social stigmatization due to confusion with leprosy.[1]

Differential diagnosis

- Leprosy
- Pityriasis versicolor
- Pityriasis alba
- Onchocerciasis
- "Bleaching" practices like the misuse of potent topical corticosteroids as adjuncts with hydroquinone
- Lichen sclerosus

Management

- Therapeutic treatments are not yet available. Proper explanation and reassurance of the patient is important. Good and practical advice about sun protection and local camouflage can often decrease the psychological burden of the disease a lot.
- Topical treatment (for small localized areas) with intermittent potent corticosteroids during a set period of time, eg four days a week for 6 months, in combination with controlled UV exposure.
- If available topical calcineurin inhibitors (TCI) like tacrolimus (0.03% and 0.1% ointment) or pimecrolimus (1% cream) may be used. The advantage is that they don't cause cutaneous atrophy.[8]
- Sometimes dapson can be useful. Dosage: 1-2mg/kg daily.
- UVB 311 nm phototherapy can be given in a specialized centre.

Clinical picture

Round, oval shaped white macula on the face

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Fixed drug eruption

Epidemiology

Fixed drug eruption (FDE) is one of the most common types of drug eruption among children presenting in (dermatology) clinics.[1;2] The incidence of fixed drug eruptions among both children and adults in several studies from Asia varied between 1 to 9%.[3;4] In recent studies from Nigeria, the hospital based prevalence of drug eruptions was 1% of which half was caused by FDE. [5;6]

Etiology and pathogenesis

Lesions develop up to several weeks after first exposure to the causative drug but may develop within 24 hours after subsequent exposures. Although the pathogenesis remains unclear positive patch test results suggest type IV hypersensitivity. Patch test results vary greatly, depending on the causative drugs.[1;2;7] The drugs most frequently associated with FDE are barbiturates, paracetamol, sulphonamides (e.g. trimethoprim-sulfamethoxazole), anti malarials and various other antibiotics, especially tetracyclines. FDE caused by Sulfa-based antimalarials frequently affect the face, lips, and limbs, whereas co-trimoxazole frequently causes genital and oral lesions. [1;6;8]

Clinical findings

The characteristic finding in FDE is recurrence of the lesions at the same sites. Lesions are sharply demarcated round or oval erythematous to violaceous / black plaques 2 to 10 cm in diameter. Usually they present as a single lesion or in limited numbers and are localized. Any cutaneous or mucosal surface can be involved including lips and genitals. With repeated episodes, the lesions may increase in size and/or number and present with more profound hyperpigmentation.[1;2;6;8]

Differential diagnosis

- Insect bites
- Urticaria
- Erythema multiforme

Management

- Prevent recurrence by identification of the responsible drug.
- Counsel the parents / caretakers about proper drug use and avoidance of responsible drugs.
- When itchy : Calamine lotion to apply 2 times daily.
- For severe itchiness oral antihistamines like piriton or promethazine can be used. For the dosages see urticaria.

Clinical pictures

Sharply demarcated, round lesions with a hyper pigmented centre and an erythematous edge

Detail, round lesion with a hyperpigmented centre

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Keloids

Epidemiology

For centuries, keloids have been a well known clinical problem and despite considerable research to unravel this phenomenon there is still no universally accepted or effective treatment.[1] Keloids and hypertrophic scars occur worldwide in all skin types but they are more common in people of African descent. Incidence rates of 16% among adult Africans have been reported while these percentages were considerably lower among schoolchildren.[2;3] In severe forms they can become disabling.

Etiology and pathogenesis

Hypertrophic scars and keloids are formed from excessive scar tissue formation at the site of prior skin injury. There is often a familial tendency for developing hypertrophic

scars and keloids but the pathogenesis remains unknown. Most probably nutritional, biochemical, immunological, and genetic factors play a role in the abnormal wound healing.[1;4;5] Another hypothesis is the influence of change in hormonal status. This might be the reason that in children before puberty there is no keloid formation after piercing the earlobes. Unfortunately prevention is often not successful.

Clinical findings

Keloids are fibrous tumors caused by overgrowth of connective tissue. They occur as a result of skin injury, such as burns, surgical or tribal cuts and ear piercing but also after inflammatory skin diseases like acne and herpes zoster.

Sites of predilection are shoulders, upper back, chest and earlobes. At first lesions are pink-to purple and often pruritic and painful. Hypertrophic scarring is restricted to the area of the original lesion and has a tendency of gradual resolution over time. Keloids can migrate into adjacent tissue to form hard, irregular shiny ridges or plaques and are persistent. [4;6]

Differential diagnosis

- Differentiating between a hypertrophic scar and keloid can be difficult.
- Scleroderma
- Dermatofibroma
- Kaposi sarcoma

Management

- Keloids and hypertrophic scars are chronic skin conditions, their treatment also takes time!
- One of the most important things that one can do to prevent the formation of keloids is to avoid trauma to the skin, attend to cuts or abrasions immediately to minimize inflammation and infection, avoid ear piercing and refrain from elective surgery unless medically indicated.
- Intralesional steroid injections: eg kenacort (1:40) on a 1:1 dilution with lidocain 2% once every 3 weeks.
- The following treatments should be preferably carried out in a specialized or university hospital:
  - ➢ Surgical excision of keloids leads to recurrence and more deformity. In severe cases debulking may be needed, and should be followed by regular intralesional steroid injections (7)
  - ➢ Cryosurgery in combination with intralesional corticosteroids can be used for small lesions.
  - ➢ Pressure with silastic gel sheets or pressure garments at night for several months.
  - ➢ Radiotherapy is highly successful but the use is limited due to its damaging long term side effects

Clinical picture

Keloid formation in a 14 year old girl after ear piercing

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Urticaria

Epidemiology

Urticaria is seen in 1-5% of the population and may present at any age. There are no known racial differences. It is more common among women with a female: male ratio of 2:1.[1-3]

Etiology and pathogenesis

Urticaria is a vascular reaction of the skin characterized by mast cell degranulation In children they caused by several factors like allergic or hypersensitivity reactions food (fish, milk, tomatoes, citrus fruits, cocoa, strawberries), drugs (aspirin, pethidine, hydralazine, ibuprofen) insect bites (bee, wasp, mosquito). [4]Also viral infections, mycotic infections, helminthic infections and skin contact with allergens can be a cause. Physical urticaria may be induced by cold, heat, pressure and exercise. In the majority of the cases, the cause remains unidentified.[5]

Clinical findings

Urticaria are well demarcated small (< 1 cm) to large (> 8cm) smooth, slightly elevated patches (wheals) which can itch severely. Individual lesions are self-limiting and resolve

in several hours but may be recurrent over weeks. Chronic urticaria is defined as urticaria with episodes lasting longer than 6 weeks.[6]
They are erythematous or white with an erythematous rim. The erythema which may be prominent in a light skin is not visible on a dark skin. Lesions can be oval, annular or serpiginous. They can appear anywhere and at any interval on the body and as angioedema in the face. The lesions are prutitic. Some patients also show dermographism. Normally urticaria in children is an isolated event, a massive reaction may occur which can lead to an anaphylactic shock.[5;7;8]

Differential diagnosis

- Contact dermatitis
- Maculopapular drug eruptions
- Insect bites
- Pityriasis rosea
- Leprosy reactions

Management [6;8;9]

- Avoid or treat the cause if possible. A thorough history is essential.The treatment depends on the severity, the duration and the type of hives.
- Further investigations, unless aimed at a specific suspected cause, are usually negative and not helpful. Only in debilitating chronic urticaria the following may be considered: Blood count, liver function test, kidney function test, infection parameters, allergy test and tests for autoimmune diseases.
- The most common treatment is oral antihistamines which controls the itching. Wheals may still be visible.

Sedating antihistaminica

*__Piriton (chlorphenamine maleate)__ (British National Formulary)

- Child under 1 year not recommended.
- Child 1-2 years: 1 mg twice daily.
  Oral solution (Syrup, chlorphenamine, 2mg/5mL) 2.5ml twice daily.
- Child 2-5 years: 1 mg 4 to 6 times daily, maximum 6 mg daily.
  Oral solution (Syrup, chlorphenamine, 2mg/5mL) 2.5ml 4 times daily.
- Child 6-12 years: 2 mg 4 to 6 times daily, maximum 12 mg daily.
  Tablets (chlorphenamine, 4 mg) ½ tablet 4 times daily.
- Above 12 years: 4 mg 4 to 6 times daily, maximum 24 mg daily.
  Tablets (chlorphenamine), 4 mg) 1 tablet 4 times daily.

*__Phenergan (promethazine)__ (British National Formulary)

- Child under 2 years not recommended.
- Child 2-5 years : 5-15 mg daily in 1-2 divided doses.
  Oral solution (Syrup, promethazine, 5mg/5mL) 5-15ml daily in 1-2 divided doses.
- Child 5-10 years: 10-25 mg daily in 1-2½ divided doses.
  Tablets (promethazine 10 mg) 1- 2½ tablet in 1-2 divided doses.
- Above 10 years: 25 mg at night, increased to 25 mg twice daily if necessary.
  Tablets (promethazine 25 mg) 1 tablet 2 times daily.

Non-sedating antihistaminica

*__Cetirizine (cetirizine)__ (British National Formulary)

- Child under 2 years not recommended.
- Child 2-6 years: 5 mg daily or 2.5 mg twice daily.
  Oral solution (Syrup, cetirizine hydrochloride, 5mg/5mL) 5 mL daily or 2.5ml twice daily.
- Child over 6 years: 10 mg daily or 5 mg twice daily.
  Tablets (cetirizine hydrochloride 10mg) 1 tablet daily or ½ tablet twice daily.

- If the first antihistamine is not effective, it might be necessary to increase the dose, or use a different antihistamine. Sometimes a combination of antihistamines is effective.
- In case of dermographism a combination of H1 (like Piriton) and H2 (like cimetidine) antihistamines is advisable.
- Oral steroids (prednisolon) in moderate dose for a few days can be helpful in severe cases of acute hives. They are not recommended long term because of adverse effects. Topical steroids like betamethason cream might be used twice daily for a short period in the case of severe itching.
- Avoid the use of aspirin, codeine and nonsteroidal anti-inflammatory drugs like ibuprofen.

Clinical pictures

Urticarial wheals on the back of an 18 year old girl

Dermographism on an forearm

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Papular urticaria

Epidemiology

Papular urticaria is regularly seen among schoolchildren in sub-Sahara Africa, especially in countries with a hot and humid climate.[1-3] The prevalence rate in Europe and the USA is unknown but it tends to be more evident during spring and summer months.[4] Papular urticaria are mainly seen among children between the age of 2 and 12.

Etiology and pathogenesis

Papular urticaria is a hypersensitive reaction to contact with arthropods, especially insects such as mosquitoes, fleas, mites, flies and bedbugs.[4;5] A type I hypersensitivity reaction plays a role in the pathogenesis of papular urticaria but delayed type (type IV) reactions are more important. Children eventually outgrow this disease, probably through desensitization. There may be a relation with atopy and poverty.

Clinical findings

The classic presentation of papular urticaria includes crops recurrent pruritic papules and papulovesicles and varying degrees of local edema. Individual papules may surround a wheal and display a central point. Scratching causes erosions and ulcerations, so secondary pyoderma is common.[8;9]

Differential diagnosis

- Insect bites
- Impetigo
- Scabies

- Dermatitis herpetiformis
- Papular pruritic rash (in HIV infection)

Management

Prevention: use insect repellents and impregnated bed nets.

- Mild topical steroids like hydrocortisone 1% two times daily.
- Topical antipruritics such as calamine lotion. Gels or lotions containing menthol or camphor may also be used sparingly in children. Do not use in infants.
- Systemic sedating antihistamines like piriton or promethazine can be tried for relief of the itching. For dosages see urticaria.
- In case of a secondary infection oral antibiotics like cloxacillin or erythromycin can be given.For dosages see impetigo.

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Clinical pictures

Severe itching papules in a Ghanaian schoolboy…

Papular urticaria in a young child

Skin conditions

Keratosis pilaris

Epidemiology

Keratosis pilaris is a common and harmless condition of keratinized hair follicles, especially among children.[1;2] It can also be seen as a symptom of the skin disease ichthyosis vulgaris and considered a symptom of atopy. It is more common in people who have a dry skin, or who have eczema.[3]In the USA between 50-80% of children, the majority of them female, are affected. Among schoolchildren in sub-Sahara Africa and other parts of the world these numbers are much lower.[2;4-6]

Etiology and pathogenesis

This disorder is characterized by grouped, horny, keratotic follicular papules predominantly located on the extensor surfaces of the proximal limbs, the posterolateral upper arms and anterior thighs. It is usually asymptomatic, sometimes slightly itchy especially when the skin is dry, and it may be disturbing cosmetically. Treatment is marginally effective and only provides temporary relief. The cause is unknown but there is hyperkeratinization which is partly inherited. This skin condition seems to run in families, which is consistent with autosomal dominant transmission. Ichthyosis vulgaris is caused by a mutation in the filaggrin gen and there is a close relationship with dry skin, allergies and eczema.[7-

Clinical findings

Numerous small, rough papules around hair follicles on the upper arms, legs, and buttocks can be seen, leading to a "chicken skin" appearance. Inflammation can be present and scratching can cause secondary infection. In the dark skin it often leads to hyperpigmentation around the follicle. Keratosis pilaris tends to fade slowly with age.[1;8]

Differential diagnosis

- Acne
- Milia
- Folliculitis
- Xerosis cutis
- Pityriasis rubra pilaris
- Lichen planopilaris

Management

- Explain to the patient that it is a chronic skin condition and it can be a part of other skin diseases like ichthyosis vulgaris. Improvement often takes months and the bumps are likely to come back.
- To prevent excessive dryness the skin should be treated regularly with an emollient cream or ointment like aqueous cream, emulsifying ointment, creams or ointments containing lactic acid 5%, coco butter or shea butter.
- Topical treatment with keratolytic ointments 3%-5% salicylic acid or ureum in the same dosage. In case of inflammation topical mild / moderate steroid ointments like hydrocortisone 1% or triamcinolon acetonide 0.1% can be used two times daily.
- Scrubbing the skin, eg. with a pumice stone.

Clinical picture

Hyperkeratotic papules…..."Chicken skin"

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Xerosis Cutis / Dry skin

Epidemiology

A very dry skin xerosis cutis or asteatosis cutis has been seen in several studies among schoolchildren in sub-Sahara Africa. Frequent washing with soap due the hot, humid climate and subsequent sweating, could explain the high prevalence.[1-4]

Etiology and pathogenesis

The etiology of xerosis cutis is multifactorial. The role of the barrier function of the stratum corneum is important. When the barrier is impaired the skin will be dry because of trans-epidermal water loss and will be more vulnerable for both infectious and inflammatory skin diseases.[5] Several studies suggest that black skin has a higher trans-epidermal water loss than light skin types.[6]

Clinical findings

Dry skin is characterized by a dull color, rough texture and elevated number of ridges.[7;8] Dry skin often itches and could lead to prurigo simplex and eventually to secondary infection; it can also trigger or worsen eczema.

Differential diagnosis

- Allergic contact dermatitis
- Nummular dermatitis
- Scabies

Management

- The use of soap and the frequency of washing should be reduced. The skin should be treated twice daily with an emollient cream or ointment like aqueous cream, emulsifying ointment, coco butter or shea butter.

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Clinical picture

Dry, rough skin with some superficial cracking (elevated ridges)

Pityriasis alba

Epidemiology

Pityriasis alba occurs mainly in infants, children and adolescents and is more often diagnosed among children with a darker complexion but may occur in individuals of all skin types.[1] It is seen more frequently among male than female and among eczema patients.[2] Prevalences of 8.4 % in India, 5,4 % in Ethiopia and 13.1 % (among children with eczema) in Nigeria have been published.[3-5]

Etiology and pathogenesis

The etiology and pathogenesis are still poorly understood. Recent studies have found direct correlations between the incidence of pityriasis alba and atopy, the amount of sun exposure, and the frequency of bathing. Because it is usually asymptomatic, findings are often incidental. Without intervention, the lesions can persist for months to years and the hypo pigmentation usually does not clear with steroids but will clear in time.[6;7] There is no difference in the number of melanocytes between lesional and normal skin which can be of help when diagnosing and differentiating pityriasis alba from other skin disorders with hypo pigmentation.[1]

Clinical findings

Pityriasis alba is a skin disorder characterized by asymptomatic, hypo pigmented, slightly scaling patches with unclear margins. It is one of the minor features of eczema and is primarily seen on the face and the trunk. Although treatment with emollients and mild topical corticosteroids may accelerate the repigmentation, they have limited efficacy.

Differential diagnosis

- Leprosy
- Vitiligo
- Pityriasis versicolor

Management

- Explain that the condition is not serious and will disappear in time.
- The skin can be treated regularly with an emollient cream or ointment like aqueous cream, coco butter or shea butter.
- Apply a mild topical corticosteroid cream like hydrocortisone 1% in case of inflammation.
- If available topical calcineurin inhibitors (TCI) like tacrolimus (0.03% and 0.1% ointment) or pimecrolimus (1% cream) may be used. The advantage is that they don't cause cutaneous atrophy.[2]

Clinical pictures

Hypopigmented macules on the trunk

Hypopigmented macules in a patient with eczema

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Chapter 9

General Discussion

Summary and concluding remarks

General Discussion

The aims of this thesis were to measure the prevalence of different skin diseases among schoolchildren in three sub-Saharan African countries, Ghana, Gabon and Rwanda. Population-based prevalence figures are needed for reliable planning of national health and prevention programmes but are scarce in Africa because of the laborious and thus expensive way to achieve them. We were especially interested in the differences of the prevalence between schools in rural and urban areas and between schools with a different socio economic level (SEL) based on school fees paid.

Because of the high prevalence of tinea capitis we analyzed the causative agents in the Ghana 1 and Gabon studies. We also made a summary of other cross-sectional studies restricted to tinea capitis in schoolchildren and carried out in other sub-Saharan countries (Table 3 **Chapter 4**).

Besides the point-prevalence we also estimated the period-prevalence and possible risk factors of eczema in a case control study. During our research in Ghana we unexpectedly discovered a significantly higher prevalence of acne vulgaris in urban areas as compared to rural areas, after which we decided to analyze the possible risk factors.

If we want to improve the quality and availability of skin disease treatment in Africa, community based studies like our study are very important.[1-3]

Although we got a good impression of the prevalence of skin diseases among schoolchildren we didn't investigate the disease burden. In future studies it would be interesting to assess the impact of skin disease on the quality of life in comparison with that of chronic nondermatological diseases. Several studies like this have been carried out in the industrialized world but they are rare in developing countries.[4]

The outcomes of the different studies in relation to the literature are discussed in the following sections.

The prevalence of skin diseases among schoolchildren in Africa

Not many population based studies about skin diseases among schoolchildren have been conducted in Africa.[5-7] Population based prevalence studies are important in order to plan health care services.[8-10] The strength of our population based study is the large number of schoolchildren investigated; the research was conducted by a dermatologist or a team of dermatologists; and it was performed in three different countries in different regions in Africa (**Chapter** 2). We separated skin diseases and skin conditions as different entities, because several skin conditions like dry skin can be indicative of skin disease but not necessarily so. In other epidemiological studies skin conditions like keratosis pilaris and xerosis cutis are counted as skin diseases and increase the overall prevalence rates. The found prevalences in our study are figures of real skin diseases and can now be set against other epidemiological studies to allow a better comparison with other past and future studies. Measuring a point-prevalence, as we did

in our studies, creates a chance of underestimation especially when chronic, relapsing diseases like eczema are involved.[6;11] Also in this respect our figures can be considered as conservative.

The prevalences of one or more skin diseases per child were high: 34.6% and 42% in both Ghana studies and 45.8% and 26.7% in the Gabon and Rwanda studies. For the percentages of infectious and inflammatory diseases together the prevalences were 22% and 26% for both Ghana studies and 37% and 27% in the Gabon and Rwanda studies. In our study the majority of skin diseases found were skin infections.[11] (figure 2 **Chapter 2**)

In a review of prevalence studies of skin diseases in children in sub-Sahara Africa, the prevalences were ranging from 21% to 87%.[12] The major causes of infection in our studies were tinea capitis and pyoderma, this pattern was conform other African studies. The point prevalence of any skin disease in a recent study among schoolchildren from Dar es Salaam (urban environment) in Tanzania was 57% which was comparable with a study from rural Tanzania which showed a prevalence rate of 55%.[6;8] The point-prevalence of infectious skin diseases in the latter study was 30.4% which was comparable with our studies and other studies from African countries like Nigeria, Ethiopia and Kenya.[5-7;13] Also among schoolchildren in other parts of the world, living in poor conditions, the same pattern can be observed.[14;15] Climatic conditions as heat and humidity, overcrowding, malnutrition and other aspects of poverty and negligence can lead to high figures of infectious diseases in the developing world.[12]

The percentages of bacterial infections in our studies were higher in both Ghanaian studies, most probably due to climatic factors like heat and humidity. Pyoderma was the second cause of skin infection. Among pyoderma we considered bacterial skin diseases like impetigo, folliculitis, abscesses, furuncles and carbuncles. In other studies performed in Africa like in Ethiopia, Mali and Tanzania percentages are mentioned between 5.6% and 4%.[5;6;16-18] In one study from Kenya[10] and one from India[14] higher prevalences of 12,7% and 11.4% have been found. In our studies we found a low prevalence of scabies and other infestations like pediculosis capitis. In several other studies prevalence rates between 0.7% and 30.5% are mentioned.[5;6;19-21] A recent review commented upon this considerable differences in prevalence and stated that scabies is quite common in developing countries but its distribution can be subject to a cycle of infection and can also have considerable regional differences.[22] Since dermatologists in our study checked all suspect cases using direct microscopy of material mounted in 20% potassium hydroxide it is not likely that we missed many.

High prevalences of infectious skin diseases as seen in our study and other studies from developing countries can be seen as a sign of poverty and negligence. Although most skin infections are not life threatening and have a lower mortality rate compared with other diseases they can damage the skin and harm the health and wellbeing of children infected.

The prevalence and causative organisms of tinea capitis in Africa

Infections with tinea capitis are endemic among schoolchildren in sub-Sahara-Africa. Tinea capitis is very contagious especially within big families and when there is overcrowding at schools. In several other studies performed in Africa prevalences were found of 29% in Zimbabwe[23], 9.8% in Mozambique[24], and 9.4% in Nigeria[25] (table 3 **Chapter 4**)

In our study there were quite some differences between the different prevalences in tinea capitis (8% and 9% in both Ghana studies and 26% and 21% in Gabon and Rwanda). One of the reasons that the prevalences in the Ghana studies were lower than in Gabon and Rwanda may be that both Ghanaian studies also included schools with a high or middle SEL where prevalences of tinea capitis were much lower. A lower SEL and the consequent overcrowding appear to be a major risk for tinea capitis (**Chapters 3 and 4)**. In several studies from the industrialized world much lower prevalences for tinea capitis are found.[26-29] Apparently, the prevalence rate of tinea capitis is going down rapidly as soon as the SEL increases.

Prevalence figures of clinically positive tinea capitis in Ghana, Gabon and Rwanda

	Ghana 2004	Ghana 2007	Gabon 2005	Rwanda 2007
Number of children	463	1394	454	2528
T.capitis	39 (**8.4**)	121 (**8.7**)	105 (**23.1**)	522 (**20.6**)
Rural	30 (**13.3**)	79 (**10.5**)	55 (**26.3**)	318 (**21.9**)
Urban	9 (**3.8**)	42 (**6.6**)	50 (**20.4**)	204 (**19.0**)
Low SEL	38 /315 (**10.8**)	100 / 967 (**10.3**)		
Middle / High SEL	1 / 112 (**0.9**)	21 / 427 (**4.9**)		

Because little was known about the causative agents of tinea capitis in Ghana and Gabon we send samples from scales and hairs of clinically positive tinea capitis of the Ghana 1 and Gabon studies to the Mycology Laboratory of the Department of Dermatology of the Leiden University Medical Centre in Leiden, the Netherlands for species identification (**Chapters 3 and 4**). T. violaceum (26%) was the most prominent species in Ghana, followed by T. tonsurans (22%) and M. audouinii (15%). No T. soudanense was found while in other studies in the region this species was the most frequent causative agent. [25;30-33] Also in our study in Gabon T. soudanense (29.4%) was the most frequent causative agent followed by T. tonsurans (27.9%) and M. audouinii (25%). Although T. violaceum was the most prominent species in the Ghana study (26%) it had a low prevalence in Gabon (8.8%). In South Africa (90%), Tanzania (14.3%) and

Ethiopia (84%) much higher prevalences are found.[6;34;35] We didn't find any M. canis in Ghana and only 1.5% in Gabon which was conform other studies in Africa where anthropophilic infections are much more common than zoophilic infections. Only in recent studies from Nigeria (7.3%) and Tanzania (46.7%) M. canis had a high prevalence which is normally a pattern seen in the USA and Europe.[6;26;27;36] However, also high prevalences of T. tonsurans can be found in the western world due to immigration patterns.[26;27;29;37]

Which species is causing tinea capitis is highly dependent on geography, time and social status. In our studies the most frequently seen were anthropophilic which are rapidly spreading in circumstances of overcrowding. Tinea capitis caused by anthropophilic species can be considered as a marker of poverty.

The prevalence of eczema among schoolchildren in Africa

The prevalences of inflammatory skin diseases like acne vulgaris and eczema were considerably higher in the urban areas and in both Ghanaian studies especially in the schools with a middle/high SEL (table 3 **Chapter 5**). This is a pattern seen in industrialized countries.

Eczema is a growing problem in Africa, particularly amongst children.[13;38-42] In the last decades the prevalence of eczema in the industrialized world has increased rapidly. Period prevalences between 15% and 30% have been found in Northern Europe, North America, Japan and Australasia.[43-45] Most of these period prevalences were obtained by questionnaires adapted from the International Study of Asthma and Allergies in Childhood (ISAAC).[46;47] The main advantages of the use of questionnaires is that they are relatively easy and cheap to distribute to children and their parents / care takers and they can be used to measure disease prevalences in larger populations.[48] They measure period prevalences which are usually higher than the point- prevalences as determined by physical examination. Because eczema is a chronic relapsing disorder a period-prevalence may be the best method to determine its frequency. In our study (**Chapter 5**) we determined point-prevalences in all four studies by physical examination of all children by a dermatologist or a team of dermatologists (in total 4839 children), which is considered the Gold standard. The found prevalences were low (1.5% and 1.6% in both Ghanaian studies and 4.0% and 0.8% in the Gabon and Rwanda studies). These figures were in agreement with other studies in Africa though recent studies have shown an increase, especially among infants.[7;38;49;50] In both Ghanaian studies we also measured by questionnaire the period-prevalences which were about twofold higher than the point-prevalences (2.6% and 4.4%). These higher figures could be expected because of the chronic relapsing character of eczema. Comparing other period-prevalences in Africa, which in other sub-Saharan countries were generally more than 10%, our figures were low (table 5 **Chapter 5**). Ideally eczema among children, diagnosed at a certain point by physical examination by a dermatologist (point-prevalence) should also be detected

with the questionnaires adapted from ISAAC (period-prevalence). However, in the Ghana 1 study the sensitivity was only 33% and in the Ghana 2 study even lower, 10% (see table 4 **Chapter 5**). The use of ISAAC adapted questionnaires does not seem to be a reliable method to determine prevalence figures of eczema in an African setting. Factors related to translation of the questionnaires from the English language as well as cultural and educational differences in developing countries seem to play an important role.[48;51;52] The conclusion that in Africa ISAAC adapted questionnaires are not a reliable method to determine prevalence figures has been confirmed in another recent study performed in South Africa.[53]

The repeated screening of large groups of children by a team of dermatologists will be too expensive and give (point) prevalences which are, due to the chronic character of eczema, too low. Since the ISAAC adapted questionnaires gave reliable figures about period-prevalence of eczema in Anglophone countries, the difficulties of the translation from the English language as well as the cultural and educational differences have to be reassessed and solved in order to make it an useful tool in an African setting.[48]

Characteristics of eczema among schoolchildren in Africa

Besides the prevalences we were also interested in the allergic characteristics of African schoolchildren and the possible risk factors for developing eczema. The rising prevalence of eczema might be related to improved sanitation and reduction in childhood infections which is known as the hygiene hypothesis.[54-56] Bacterial and viral infections lead to a maturation of the immune system and reduce the expression of the pro-allergic T-helper-2 (Th2) responses. According to this hypothesis an increasing gross national per-capita income, a higher socio-economic status, reduced crowding at home, eradication of endoparasites, vaccinations and the growing urbanization in Africa are all factors which can lead to a higher prevalence of eczema (Chapter **6)**.[41;57-60]

In order to study the risk factors for eczema in an African setting we selected children with a moderate to severe eczema in three hospitals in Accra, Ghana and matched one to three controls for age and gender without any visible symptoms of eczema from the same school and class. Elevated total IgE levels were associated with eczema among the schoolchildren although also a high proportion of elevated total IgE levels was found in children without eczema. Elevated cockroach specific IgE levels appeared to be the main cause behind the association between elevated total IgE levels and eczema in our study. The association between any positive skin prick test (SPT) for house dust mite, cat, dog, grass, peanuts and eczema was less pronounced. The same pattern was seen in a study in Ethiopia where 60% of the children with clinical eczema didn't show any positive SPT.[50] There is evidence for a causal relationship between helminth infections and reduced SPT responsiveness to allergens.[61] This protective effect of helminth infections on the development of eczema could not be found in our study because only very few children in this urban population had a helminthic infection at the time of

the study. Maybe helminth infections in the past could have played a role.[60] In the same study from Ethiopia no evidence was found that the risk of eczema was reduced in the presence of 1 or more parasite infections. They found even an increased risk of eczema in the presence of any parasite infection, especially caused by Trichuris.[50] A non-significant positive association between frequent bathing and eczema was observed in our study. Frequent bathing with water and soap is considered to be a healthy exercise in Ghana. Another non-significant association with the development of eczema was found with sleeping on a mattress, an association not found in an earlier African study.[41] Sleeping on a mattress might be an indicator of a higher socio-economic status and can lead to higher exposure to house dust mite and cockroach antigens which all influence the occurrence of eczema.

Important factors in the development of eczema in individuals from European and Asian descent are mutations in the filaggrin gene (FLG), a key protein involved in maintaining skin barrier function and hydration.[62] Only recently research to this has been carried out in Africa.[63] It would be very interesting to look for mutations in the FLG in our study. Plans for this have been made.

Prevalence and risk factors of acne vulgaris in Africa

Acne vulgaris affects between 30% and 95% of the adolescents in the industrialized countries.[64-66] The prevalence is considerably lower in developing countries.[5;6;14] In our studies (**Chapter 7**) the prevalences of acne vulgaris were 3.2% and 4.7% in both Ghanaian studies and 1.1% and 1.3% in Gabon and Rwanda. The prevalences in Gabon and Rwanda were low comparing with the both Ghanaian studies. One of the reasons might be that in both Ghanaian studies also urban private schools with a higher SEL were involved. In **Chapter 7** we studied especially the Ghana study 2 to look at the difference between the prevalence rates among rural and urban schoolchildren and possible risk factors. There are several hospital-based studies about the prevalence of acne vulgaris in Africa, [17;67;68] but only a few community based studies that compared prevalences of acne among schoolchildren in rural and urban areas.[5;69] In table 1 (**Chapter 7**) the prevalence of acne vulgaris in several developing countries is reported. In the Ghana 2 study the schools were categorized as public (no school fees), private (middle to high school fees) and private rich (very high school fees) to reflect the average socioeconomic level of the children, respectively low, middle to high. The prevalence of acne was less than 1% in the rural schools and varied between 1.3% in boys between 9 and 10 years old to 28.4% in girls between 13 and 14 years old in the urban schools. One of the main reasons of this difference might be the significantly lower height and weight of the rural children. An association between overweight and acne has been described before.[70;71] The observed association between a higher BMI (body mass index) and acne among the children who were attending the urban schools would fit in this hypothesis. In our study we only looked at children between 9 and 16

years. Female gender, increasing age and a high BMI were risk factors for inflammatory acne vulgaris (table 3 **Chapter** 7). The observed relation between the prevalence of acne and a higher BMI is one of the first reported in an African study. With the fast growing urbanization and westernization in developing countries, an increase of inflammatory skin diseases can be expected.

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Summary and concluding remarks

Many skin diseases among schoolchildren in sub-Sahara Africa cause disturbing complaints like itch and pain and several of them are contagious. This high prevalence causes a major public health problem. Although in several studies and also in our studies skin diseases present in large numbers, they don't get the attention they deserve. A high percentage of visits to hospitals or other health institutions are caused by skin diseases. Most of them can easily be prevented and treated.

The studies presented in **Chapter 2** focused on descriptive epidemiology to characterize and report the point-prevalence of skin diseases among schoolchildren in three African countries. We found high figures for the point-prevalence of infectious diseases like tinea capitis and pyoderma. The prevalence of the so called "typical" tropical diseases was very low. Most of the diseases are not difficult to diagnose and can easily be treated. Not many population based studies about the prevalence of skin diseases among schoolchildren in Africa have been published. The majority of studies are hospital or institution based. We have performed 4 studies in 3 different African countries which makes it quite unique. Most other studies present also high figures, especially of skin infections.
Eczema is a growing problem in sub-Sahara Africa but the point-prevalences among schoolchildren are still much lower than those found in the industrialized world. The point-prevalence of acne vulgaris was low in the rural areas and much higher in the urban schools with a higher socioeconomic level (SEL) (i.e. higher school fees).

In **Chapters 3 and 4** the high prevalences of tinea capitis among schoolchildren in Ghana and Gabon were further studied and the causing species analyzed. The prevalences were higher in rural schools with a low SEL. The most prominent causing species were anthropophilic moulds which are mainly spread under circumstances of crowding. They can be seen as poverty markers. T. soudanense was not encountered in our study in Ghana, which was an exceptional observation because in other studies from West Africa it was the main causing species.

In **Chapter 5** we focused especially on the prevalence of eczema. Most of the studies concerning the prevalence rate of eczema in Africa are hospital-based and therefore less reliable when estimating the prevalence of eczema on a national scale. The point-prevalences in our study of eczema amongst schoolchildren, as measured by physical examination by a dermatologist, were low and comparable with other studies in Africa. There was not a significant difference between rural and urban areas however, as was anticipated. We analyzed the use of ISAAC (International Studies on Allergy and Asthma) based questionnaires to determine the period-prevalence of eczema. The sensitivity

and the positive predictive values were very low in our hands, which suggest that this method might be less suitable and reliable to identify children with eczema in an African setting. Most probably these discrepancies are caused by factors related to translation of the questionnaires from the English language as well as cultural and educational differences in developing countries.

In **Chapter 6** we were looking for risk factors of eczema among schoolchildren in Accra, Ghana. Elevated total IgE levels were associated with eczema among the schoolchildren, although also a high proportion of elevated total IgE levels were found in children without eczema. Elevated cockroach specific IgE levels appeared to be the main cause behind the association between elevated total IgE levels and eczema in our study. The association between any positive SPT (skin prick test) and eczema was less pronounced. A positive association, though non-significant, between frequent bathing and eczema was observed. Another non-significant association with the development of eczema was found with sleeping on a mattress which might be an indicator of a higher socio-economic status and can lead to higher exposure to house dust mite and cockroach antigens which can all influence the occurrence of eczema.
Both the studies from **Chapter 5 and 6** contribute to increasing our understanding of epidemiology and characteristics of eczema in sub-Sahara Africa.

Chapter 7 was based on analytic epidemiology where we searched for risk factors of acne vulgaris. Acne is a growing problem in Africa although the prevalence is still much lower than in industrialized countries. In our Ghana 2 study we observed a direct positive relation between an increasing body mass index (BMI) and the prevalence of acne. The relation found between a higher BMI and the prevalence of acne vulgaris, especially among children in urban areas is of importance because of the growing problem of obesity in Africa. Because of the "westernization" in urban areas in developing countries a steep increase of the prevalence of acne vulgaris in Africa can be expected. This was one of the first observations of this kind described in Africa.

In **Chapter 8** the epidemiology, etiology and pathogenesis, clinical symptoms and the management and treatment of the most prevalent skin diseases among schoolchildren in Africa are described. Also some typical tropical diseases and skin conditions are mentioned. Although the prevalences in our studies of classical tropical diseases like leprosy or filarial lymph edema or a condition like albinism were low, the socio -economical impact of these diseases can be enormous. This list of skin diseases is far from complete and needs continuous improvements and additions. All skin diseases described will be freely accessible via the internet: www.africanskindiseases.org.
It is meant to be a practical guide for general practitioners, students and all others who are working with children in the medical field.

In the concluding chapter (**chapter 9**), the descriptive epidemiological aspects as well as the analysis of the causing species in tinea capitis are discussed together with the study of risk factors of eczema and acne. The different studies are discussed in view of recent findings and publications by others. The impact of statistical and methodological biases of the study design on our findings is discussed.

Concluding remarks

Our study shows that skin diseases and conditions are common among schoolchildren in sub-Sahara Africa and about one-third or more of them are affected at any given time.
The majority of skin diseases found was caused by infections and inflammation together and can be grouped into fewer than eight categories. This is important in designing training programs for medical teams involved in the delivery of health care services in sub-Sahara African countries where a big part of the population is less than 15 years of age.

Chapter **10**

Nederlandse samenvatting

List of publications

Curriculum vitae

Dankwoord | Acknowledgements

Gutta cavat lapidem, non vi sed saepe cadendo.

The drop hollows out the stone by frequent dropping, not by force; constant persistence gains the end.

Nederlandse samenvatting

De prevalentie van huidziekten onder schoolkinderen in sub-Sahara Afrika is erg hoog en vormt een belangrijk maar vaak onderschat probleem voor de publieke gezondheidszorg. Diverse huidziekten veroorzaken klachten zoals pijn en jeuk en kunnen bijzonder besmettelijk zijn. Een hoog percentage van de bezoeken aan ziekenhuizen en klinieken wordt veroorzaakt door huidziekten waarvan de meeste gemakkelijk voorkomen en behandeld kunnen worden.

In **hoofdstuk 2** worden de puntprevalenties van huidziekten onder schoolkinderen in drie Afrikaanse landen (Gabon, Ghana en Rwanda) besproken. Opvallend zijn de buitengewoon hoge percentages van besmettelijke ziekten zoals tinea capitis en pyoderma terwijl de percentages van de zogenaamde klassieke tropische aandoeningen erg laag waren. Eczeem is een groeiend probleem in sub-Sahara Afrika hoewel de puntprevalenties onder kinderen nog steeds veel lager zijn dan die in de geindustrialiseerde landen. De gevonden punt-prevalentie van acne vulgaris was laag in de plattelands (rurale) gebieden maar veel hoger in de stedelijke (urbane) gebieden, vooral op scholen met een hoger sociaal economisch niveau (hoger schoolgeld).
Er zijn niet veel bevolkingsonderzoeken beschreven over de prevalentie van huidziekten onder schoolkinderen in Afrika. De meeste onderzoeken worden gedaan vanuit ziekenhuizen of andere instituten. Dat er vier onderzoeken verricht zijn in drie verschillende landen en met zulke grote aantallen kinderen, maakt deze studie bijzonder.

In **hoofdstukken 3 en 4** worden de hoge prevalenties van tinea capitis onder schoolkinderen in Ghana en Gabon beschreven. Gedurende deze studies werd van klinisch verdachte kinderen materiaal afgenomen en in het mycologisch laboratorium op de poli Huidziekten van het Leids Universitair Medisch Centrum geanalyseerd. De gevonden prevalenties waren het hoogst op rurale scholen met een lager sociaal economisch niveau. De meest voorkomende verwekkers waren anthropophiele mycosen die vooral gezien worden op plaatsen waar mensen dicht op elkaar leven (grote klassen, gezinnen) in veelal armoedige omstandigheden. T. soudanense werd niet gevonden in de studie uit Ghana, hetgeen opmerkelijk is daar dit in de meeste studies uit West Afrika de grootste veroorzaker van tinea capitis betreft.

In **hoofdstuk 5** wordt vooral de prevalentie van eczeem beschreven. De meeste prevalentiestudies in Afrika zijn vanuit ziekenhuizen of andere instituten verricht en daarom minder geschikt om een betrouwbare schatting te doen naar de prevalentie van eczeem op nationaal niveau. De puntprevalenties in onze studie, zoals bepaald na lichamelijk onderzoek door een dermatoloog (de "gouden" standaard), waren laag en vergelijkbaar met andere studies in Afrika. De verschillen in prevalentie tussen rurale en

urbane gebieden waren niet significant. Het gebruik van op ISAAC (International Studies on Allergy and Asthma) gebaseerde enquetes werd geanalyseerd, met name om de periode prevalentie van eczeem te bepalen. De sensitiviteit en de positief voorspellende waarden waren zeer laag. Deze verschillen worden vermoedelijk veroorzaakt door problemen met de vertaling van de enquetes uit het Engels en door cultuurverschillen. Dit maakt deze methode minder geschikt en betrouwbaar om de prevalentie van eczeem bij kinderen in Afrika te bepalen.

In **hoofdstuk 6** wordt nader ingegaan op de risicofactoren voor het ontwikkelen van eczeem in Accra, Ghana. In deze studie werd een verband gevonden tussen een verhoogd totaal IgE en eczeem onder schoolkinderen hoewel er ook bij diverse kinderen een verhoogd IgE werd gevonden, zonder dat er sprake was van eczeem. Een verhoogd specifiek IgE tegen kakkerlakken leek de hoofdoorzaak te zijn van de associatie tussen een verhoogd totaal IgE en eczeem in onze studie. De associatie tussen een positieve huidpriktest op kakkerlakken antigeen en eczeem was minder duidelijk.
Een positieve associatie, hoewel niet significant, werd gevonden tussen veelvuldig wassen en het ontstaan van eczeem. Een andere, niet significante, associatie voor de ontwikkeling van eczeem werd gevonden met het slapen op een matras. Dit laatste kan wijzen op een hoger sociaal economische niveau en op een verhoging van blootstelling aan huisstofmijt en kakkerlakantigenen.Alle genoemde factoren kunnen het voorkomen en ontstaan van eczeem beinvloeden.
Beide studies zoals beschreven in hoofdstuk 5 en 6 hebben geleid tot meer kennis over de epidemiologie en de oorzaken van eczeem in sub-Sahara Afrika.

In **hoofdstuk 7** worden de risicofactoren voor acne vulgaris nader geanalyseerd. Acne is in toenemende mate een probleem in Afrika hoewel de prevalentie een stuk lager is dan in geïndustrialiseerde landen. In onze tweede Ghanese studie zagen wij een direct positief verband tussen een verhoogde BMI (Body Mass Index) en de prevalentie van acne. De gevonden relatie tussen een hogere BMI en de prevalentie van acne vulgaris, speciaal bij kinderen in stedelijke gebieden, is door het toenemende probleem van obesitas in Afrika, belangrijk. Gezien de toenemend westerse leefstijl in de stedelijke gebieden in ontwikkelingslanden kan een scherpe stijging van de prevalentie van acne in Afrika verwacht worden.
Deze studie vormt één van de eerste observaties die over de relatie tussen BMI en de prevalentie van acne in Afrika beschreven zijn.

In **hoofdstuk 8** worden de epidemiologie, etiologie en pathogenese, klinische symptomen en de behandeling van veel voorkomende huidaandoeningen onder schoolkinderen beschreven. Tevens worden ook enige" typisch" tropische ziekten besproken. Hoewel de prevalentie van klassiek tropische ziekten zoals lepra, Buruli ulcus en filariasis in onze

studies zeer laag was, worden er toch enige hiervan gepresenteerd. De redenen hiervan zijn de grote sociale en economische consequenties van deze ziekten. Dit laatste geldt ook voor een huidaandoening als albinisme.
Dit hoofdstuk is bedoeld als een praktische kinderdermatologische handleiding voor artsen, clinical assistants, verpleegkundigen, studenten en iedereen die werkzaam is in de gezondheidszorg in Afrika. Het is aanvullend op het boek: " Common skindiseases in Africa. An illustrated guide" door Colette van Hees en Ben Naafs. De lijst met dermatologische aandoeningen is nog niet compleet en dient voordurend verbeterd, aangevuld en aangepast te worden. De informatie en klinische foto's zullen vrij beschikbaar zijn op het internet via de website: www.africanskindiseases.org.

Hoofdstuk 9 geeft een samenvatting van de resultaten en bevindingen zoals beschreven in de voorgaande hoofdstukken. De verschillende studies worden vergeleken met recente bevindingen en publicaties verricht door anderen. Tevens worden methodologische en statistische tekortkomingen van de studies besproken.

Conclusies

Onze studies laten zien dat huidziekten en aandoeningen onder schoolkinderen veel in sub-Sahara Afrika voorkomen.Ongeveer een derde van de kinderen is op een gegeven moment aangedaan.
De meerderheid van de huidziekten werd gevormd door infectieuze en inflammatoire aandoeningen welke in minder dan acht categorieën samengevat kunnen worden. Dit is een belangrijk gegeven voor het maken van trainings- en behandelings programma's voor diegenen die actief zijn in de gezondheidszorg in sub-Sahara Afrika, waar het grootste deel van de bevolking veelal jonger is dan 15 jaar. Er is een grote behoefte aan gestandaardizeerde adviezen over de behandeling van de belangrijkste huidaandoeningen en aan speciale trainingen voor gezondheidszorgmedewerkers.

Publications

1 Skin diseases among schoolchildren in Ghana, Gabon and Rwanda. **Hogewoning AA**, Amoah AS, Bouwes Bavinck JN, Yazdanbakhsh M, Adegnika AA, De Smedt SK, Willemze R, Boakye DA, Lavrijsen AP
August 2012, Accepted for publication in the *International Journal of Dermatology*

2 Point and period prevalences of eczema in rural and urban schoolchildren in Ghana, Gabon and Rwanda. **Hogewoning AA**, Bouwes Bavinck JN, Amoah AS, Boakye DA, Yazdanbakhsh M, Kremsner PG, Adegnika AA, De Smedt SK, Willemze R, Lavrijsen AP. *J Eur Acad Dermatol Venereol. 2012 Apr; 26(4):488-94.*

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Curriculum Vitae

On 4 January, 1960 in Dordrecht, the Netherlands, Arjan Hogewoning was delivered by his father, a gynecologist in the same city for over twenty-five years. In 1979, Arjan passed his final examinations at the Johan de Witt Gymnasium in Dordrecht, to begin his own study of medicine at the Rijksuniversiteit Leiden. Under the guidance of Prof. Dr. Hugo van der Kaay and under the supervision of Dr. Hans Hogerzeil, he conducted, for his bachelor's degree, a malaria research project in Agogo Hospital, Ghana and subsequently wrote his first publication.

Living and working in Africa instilled such a lasting impression, that Arjan began his dream of pursuing a career in tropical medicine. Following his qualifying examination in 1989, his studies consisted of a two-year rotation in surgery at St. Antoniushove Hospital in Leidschendam, work in obstetrics and gynecology at St. Anna Hospital in Geldrop, and a three month course in tropical medicine at the Royal Tropical Institute in Amsterdam. In 1991 he moved to Botswana where he worked as Senior Medical Officer and later as Medical Superintendent in the Deborah Retief Memorial Hospital in Mochudi. He began his specialist training in dermatology and venereology in September 1995 at Leiden University Medical Center under the supervision of Prof. dr. Bert Jan Vermeer and Prof. dr. Wilma Bergman. Upon completion of his specialization in 2000, Arjan and his family relocated to Accra, Ghana where he started working as a dermatologist in the Korle Bu Teaching Hospital, Achimota Hospital, Tema Hospital, and the Akai House Clinic. Moving to Kigali, Rwanda in 2005, Arjan continued his work as a dermatologist in the King Feisal Hospital and taught at the National HIV/AIDS Institute (TRAC). In addition, twice a year between 2000 and 2010, he taught tropical dermatology and venereology at the Royal Tropical Institute in Amsterdam.

In 2009 he moved to the Slovak Republic where he works at the Department of Pediatric Dermatovenereology, School of Medicine, Comenius University, Bratislava. All the years in Africa and in the Slovak Republic his wife Door has been working in the brewing industry. In addition to his work as a dermatologist, Arjan also works as a part-time medical consultant for the Pharmaccess Foundation, an NGO linked with the Academic Medical Centre in Amsterdam. He regularly returns to Africa for both professional and personal reasons.

It was during his years in Ghana and Rwanda that most of the research of this thesis was performed under the supervision of co-promotor Dr. Sjan Lavrijsen and Dr. Jan Nico Bouwes Bavinck. It was the result of a close cooperation between the Departments of Dermatology (Prof. Dr. Rein Willemze) and Parasitology (Prof. Dr. Maria Yazdanbaksh) of the Leiden University Medical Centre.

Dankwoord | Acknowledgements

Gedurende de afgelopen jaren heb ik veel steun gehad van diverse mensen zonder wie dit "Afrikaanse" avontuur nooit volbracht zou zijn. Ik bedank ze hieronder maar besef me ook dat de lijst, hoewel uitvoerig, zeker niet volledig is.

Prof.dr.Bert Jan Vermeer Vaak sprak ik met hem over Afrika, het continent waar hij na zijn verblijf in Malawi altijd naar is blijven terugverlangen. Ik gedenk hem in vriendschap en met respect.

Prof.dr.Rein Willemze Beste Rein, als hoofd van de afdeling Huidziekten in het LUMC heb jij dit onderzoek financiëel als ook met je belangstelling altijd gesteund waarvoor ik je zeer erkentelijk ben.

Co-promotor Dr.Sjan Lavrijsen en Dr.Jan Nico Bouwes Bavinck Beste Sjan en Jan Nico, samen zijn jullie vanaf het begin nauw betrokken geweest met alle veldonderzoeken en de verwerking hiervan tot artikelen. Dit proefschrift was er zonder jullie inzet en steun nooit gekomen.

Prof.dr.Hector Addo, Dr.Margaret lartey, Dr.Daniel Boakye, Abena Amoah, Irene Akosua Larbi, Benedicta Obeng, Yvonne Aryeetey and Dr.Akim Adegnika Dear all, with the four research projects carried out in Ghana and Gabon, you have been very instrumental and of great assistance.

Dr.Stefan De Smedt en Yannick Fonteyne Beste Stefan en Yannick, bedankt voor de goede samenwerking in Rwanda waar jullie, naast een drukke ophthalmologische praktijk, een indrukwekkend onderzoek hebben opgezet.

Carel Staats en Liesbeth van der Raaij-Helmer Beste Carel en Liesbeth, jullie ben ik zeer erkentelijk voor de analyse van de tinea capitis samples hetgeen geleid heeft tot twee publicaties (hoofdstukken 3 en 4).

All children, parents and teachers in Ghana, Gabon and Rwanda Thanks to all the children, the parents and the good cooperation with the teachers it was possible to screen so many children in a well organized way.

Colette van Hees en Dr.Ben Naafs Beste Colette en Ben, jullie wil ik speciaal bedanken voor de assistentie bij het maken van een gids en website voor de behandeling van huidziekten bij kinderen in Afrika zoals beschreven in hoofdstuk 8.

Thomas Donker Beste Thomas, veel dank voor het organiseren en bewerken van alle foto's die gebruikt zijn in hoofdstuk 8.

Jan Willem Gratama en Bas Kist Beste paranymphen, jullie zijn altijd buitengewoon geïnteresseerd gebleven in ons doen en laten in Afrika, ik ben blij dat jullie me op de dag van de promotie bij willen staan.

Mijn ouders Lieve Papa en Mama, bedankt voor jullie onvoorwaardelijke vertrouwen in mij en de steun die jullie altijd gaven, waardoor ik mij heb kunnen ontplooien en mijn idealen nastreven.

Pieter, Anne en Benjamin Hogewoning De Afrikaanse jaren hebben ons tot een hechte clan gesmeed ! Veel dank voor al jullie interesse en steun de afgelopen jaren.
Door Plantenga Liefste Doortje, je hebt me -naast jouw drukke werkzaamheden- altijd enorm met dit Afrikaanse avontuur gestimuleerd, ik kan je hier niet dankbaar genoeg voor zijn.