Tinea capitis in the United Kingdom. A report on its diagnosis, management and prevention
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Abbreviations, medical and immunological terms

**Alopecia areata**  Hair loss, usually reversible, in sharply defined areas, usually involving the beard or scalp

**Anthropophilic infections**  Infections spread from human to human

**Antigen**  Any substance capable of inducing a specific immune response

**Arthroconidia**  A type of fungal spore typically produced by segmentation of pre-existing fungal hyphae

**Candidosis**  Infection with a fungus of the genus *Candida* that usually occurs in the skin and mucous membranes of the mouth, respiratory tract

**Carrier**  An individual without clinical signs and symptoms from whose scalp organisms can be grown in culture

**Cicatricial or scarring alopecia**  Irreversible loss of hair associated with scarring, usually on the scalp

**Corticosteroids**  Steroid hormones

**Cytokines**  Any of several regulatory proteins, such as the interleukins and lymphokines, that are released by cells of the immune system and act as intercellular mediators in the generation of an immune response

**Dermatophytes**  Filamentous fungi belonging to the genera *Trichophyton*, *Microsporum* or *Epidermophyton* causing parasitic infections of the skin, hair, or nails

**Dermatophytosis**  A fungal infection of the skin caused by a dermatophyte fungus, also known as ringworm

**Dysplastic lesions**  Lesions with abnormal cell changes

**Ectothrix infection**  A fungal parasite, such as certain dermatophytes, forming a sheath of spores on the outside of a hair as well as growing within the hair shaft

**Endothrix infection**  A dermatophyte whose growth and spore production are confined chiefly within the hair shaft

**Epilation**  The act or result of removing hair

**Epitopes**  The portion of an antigen capable of eliciting an immune response and of combining with the antibody produced to counter that response.

**Erythema**  Redness of the skin due to congestion of the capillaries

**Favus**  A severe type of chronic ringworm of the scalp and nails that is caused by various dermatophytes and occurs in humans and certain animals.

**γ-interferon**  A cytokine produced by macrophages and T cells that is involved in regulation of the immune system and activation of phagocytes

**Hyphae**  Long, branching filaments found primarily in fungi

**IgE**  Immunoglobulin E (typically involved in inflammatory responses)

**IgG**  Immunoglobulin G

**Il4**  Interleukin 4 (type of cytokine that acts to stimulate, regulate, or modulate lymphocytes such as T cells)
<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
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<tr>
<td>Kerion</td>
<td>Highly inflamed lesion caused by a dermatophyte fungus invading the hair follicles and marked by raised, usually pus-filled and spongy lesions</td>
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<tr>
<td>KOH</td>
<td>Potassium hydroxide</td>
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<td>Lymphadenopathy</td>
<td>Disease or swelling of the lymph nodes</td>
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<tr>
<td>Lesions</td>
<td>An infected or diseased patch of skin</td>
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<tr>
<td>Macrophages</td>
<td>A phagocytic cell of the immune system which ingests foreign material and act as antigen presenting cells and produce cytokines</td>
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<tr>
<td>MCLFA</td>
<td>Medium chain length fatty acids produced in the sebum</td>
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<tr>
<td>Onychomycosis</td>
<td>A fungal infection of the fingernails or toenails that results in thickening, roughness, and splitting of the nails</td>
</tr>
<tr>
<td>Pityriasis amiantacea</td>
<td>A condition of the scalp characterised by thick, yellow-white scales densely coating the scalp skin</td>
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<tr>
<td>Polymorphonuclear leucocytes (PMN)</td>
<td>Type of phagocytic white blood cell involved in the innate immune response to an acute inflammatory episode</td>
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<tr>
<td>Psoriasis</td>
<td>A noncontagious inflammatory skin disease characterized by recurring reddish patches covered with silvery scales</td>
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<tr>
<td>Pustule</td>
<td>A small, raised pus-containing lesion of the skin</td>
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<tr>
<td>Radiodermatitis</td>
<td>Dermatitis due to exposure to ionizing radiation</td>
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<tr>
<td>Scutulum</td>
<td>The characteristic lesion of favus, appearing as a yellow saucer-shaped crust made up of hyphae, spores and cell debris</td>
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<tr>
<td>Seborrhoeic dermatitis</td>
<td>Inflammatory and scaly skin disease affecting the scalp, face or anterior chest thought to be caused by a reaction to colonising skin surface yeasts of the genus Malassezia</td>
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<tr>
<td>Sebum</td>
<td>The semifluid secretion of the sebaceous glands,</td>
</tr>
<tr>
<td>Squalene</td>
<td>Compound found in human sebum</td>
</tr>
<tr>
<td>Stratum corneum</td>
<td>Outermost layer of the skin or epidermis</td>
</tr>
<tr>
<td>TH cells (1&amp;2)</td>
<td>T helper cells. Type of white blood cell that has various roles in the immune system, including the identification of specific foreign antigens in the body and the activation and deactivation of other immune cells.</td>
</tr>
<tr>
<td>TH1 response</td>
<td>Immune response whose main feature is cellular inflammatory response promoted via cytokines produced by TH1 cells</td>
</tr>
<tr>
<td>TH2 response</td>
<td>Immune response whose main feature is antibody production promoted via TH2 cells</td>
</tr>
<tr>
<td>Tinea corporis</td>
<td>Ringworm of the body (fungal infection) characterized by a pink to red rash and often considerable itching</td>
</tr>
<tr>
<td>Tinea capitis</td>
<td>Ringworm of the scalp (fungal infection) characterized by itchy, scaly patches and sometimes hair loss</td>
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<td>Zoophilic infections</td>
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Overview of summaries

Epidemiology

It is clear that the pattern of tinea capitis in the UK has changed in the past ten years with a significant rise in the incidence and prevalence of cases of infection due to *Trichophyton tonsurans*. This is reflected by major changes in the level of diagnostic work seen in some centres. For instance in South London the number of cases diagnosed annually at the St Johns Institute of Dermatology has increased by more than 40-fold over this period. The main focus of this epidemic has been cities where there are either long-standing or more recently established black communities. But it is clear that infection can occur in any child irrespective of their ethnic origin. It is therefore important to continue surveillance of this problem, to understand the specific needs of the ethnic minority communities, who have been the main focus of this infection, and to ensure that methods are in place to control this new outbreak of a common childhood condition.

Clinical detection

It is unreliable to depend on clinical diagnosis alone to identify cases of tinea capitis given the range of clinical expression and the high numbers of children with mild infections that are difficult to detect. Laboratory methods should be used wherever possible to confirm the diagnosis.

The carrier state

Carriage of fungi, defined as positive cultures taken by brush sampling but absence of clinical signs of infection or positive direct microscopy of hair, can occur. However in the case of *T. tonsurans* infection in some individuals it is possible to overlook limited and symptom-free infections accompanied by hair shaft invasion without highly detailed examination of the scalp.
Diagnosis

Wherever possible scalp scrapings, including hairs and hair fragments, should be used as the primary method of detection of cases. However this may be difficult to accomplish in many instances and, therefore, a second line approach would be the use of sterile brushes such as disposable toothbrushes. Cultures should be repeated after therapy. Ideally the annual diagnostic figures for tinea capitis should be collected from a number of sentinel diagnostic laboratories in order to monitor the progress of this epidemic and the effect of control measures.

Treatment of cases

For the time being it is important to recognise that there is no approved treatment for tinea capitis in childhood in the UK apart from the tablet formulation of griseofulvin.

There are a number of options:
Children with tinea capitis infections are treated with griseofulvin at a dose of at least 10 mg/kg; this may be increased to 20 mg/kg in patients with *T. tonsurans* infection or where there is failure to respond after 6 weeks of treatment. Although there is no UK approved liquid paediatric formulation of griseofulvin, it is still an effective treatment with evidence that it provides broad cover for all the different organisms that cause tinea capitis. In younger children its use may involve giving crushed tablets or suspensions of crushed tablets.

Terbinafine is now well documented as a treatment for *Trichophyton* infections, particularly those caused by *T. tonsurans*; the duration of treatment is 4 weeks. It is equivalent to griseofulvin given for 8 weeks and it is increasingly recommended as the first treatment for *T. tonsurans* infections. Its dose is doubled in *Microsporum* infections.
Itraconazole and fluconazole are alternatives, particularly in *Microsporum* infections.

The use of a topical treatment, either selenium sulphide or ketoconazole shampoo, or another topically active antifungal e.g. terbinafine cream is recommended at least twice weekly during the first two weeks of therapy and that children on treatment should not be kept off school unless their clinical condition warrants it, e.g. severe kerion. Carriers are also given a topical preparation such as selenium sulphide shampoo at least twice weekly but do not receive oral antifungals. However if there is heavy growth of dermatophytes from scalp brushes taken from children with clinically normal scalps they should be treated as if they are infected cases ie with oral therapy. Children in contact with tinea capitis should be examined very carefully for signs of infection which may be confined to a few broken hairs. If infected hairs are seen and this is confirmed by mycological examination the children should receive oral therapy.

Management of spread

There has been a change in the pattern of scalp ringworm or tinea capitis in the UK with an increased frequency of infection due to anthropophilic dermatophytes such as *T. tonsurans* that spread from child to child in the community. While the majority of cases occur in urban areas and in black children this infection can affect any child. Treatment measures depend on the use of oral antifungal therapy with griseofulvin. It is important to note, though, that the current measures for control have not been effective where spread of *T. tonsurans* has occurred elsewhere, e.g. in the USA, possibly due to the difficulty of distinguishing between carriers and children with minimal infections. *T. tonsurans* is not a new infection in the UK and in the 1970's there were outbreaks of infections in schools [46]. However control was achieved by rigorous surveillance. Although there are some differences in the new pattern of infection improving early detection rates is likely to provide some of the answers.
Introduction

Tinea capitis, or scalp ringworm, is an infection caused by dermatophyte fungi mainly found in pre-pubertal children [1]. It is characterised by infection of the hair of the scalp and scalp skin associated with symptoms and signs of inflammation and hair loss [2]. Tinea capitis is an exogenous infection caused by dermatophytes which originate from sources including other children or adults (anthropophilic), animals (zoophilic) or soil (geophilic). The main signs are scaling and hair loss but acute inflammation with erythema and pustule formation may also occur. The dermatophytes that cause tinea capitis (Table 1) can invade other parts of the body such as the nails and the body, but rarely the feet or groins. Children or adults who have neither signs nor symptoms of infection, but from whose scalps causative fungi can be grown, are described as “carriers”.

The epidemiology of tinea capitis is not static as movements of people provide the potential to introduce new species to different areas. For instance since the 1970’s there has been a progressive spread of infections caused by Trichophyton tonsurans through inner city areas of much of the USA [3] and more recently in the UK [4] and other European cities [5]. By contrast Trichophyton schoenleinii, which causes a characteristic scalp infection, favus, is becoming less common, partly because its striking clinical appearances and the tendency to scar are recognised even in remote communities. Patients with favus, or their parents, are more likely to present for treatment.

Generally the route of hair invasion in tinea capitis is thought to follow a similar pattern in all cases [2]. Spores (arthroconidia) or infected hairs are transferred by contact and by air currents onto the epidermis between follicles and adhesion occurs between the fungal spore and the stratum corneum. Thereafter the spores germinate producing chains of cells or hyphae and when contact with a hair follicle opening is made the hyphal growth is directed downwards into the hair follicle until contact with and penetration of the hair
occurs. Growth within the hair shaft appears to be unrestricted although proximal or downwards penetration (towards the hair bulb) stops at the zone of nucleated cells or Adamson’s fringe. Hyphae within the hair behave in one of three different ways depending on their survival within the shaft and the site of production of the arthroconidia. In ectothrix infections hyphae develop into chains of arthroconidia on the external surface of the hair shaft; hairs appear to be covered by a deposit and may break between 1 – 10 mm above scalp level. With endothrix infections arthroconidia are formed inside the hair shaft and the hairs may be slightly swollen but otherwise appear normal. In favus growth within the hair is not sustained and hyphae disintegrate leaving clear air-containing spaces in the hair shaft, although they survive at scalp level resulting in a tangled mass of hyphae, keratinocytes and cellular debris around hair, the scutulum.

The principal pathways of defence are the interaction between fungi and local defence mechanisms such as unsaturated transferrin, the migration of polymorphonuclear leucocytes into the area of infection and T lymphocyte activation [6]. Medium chain length fatty acids (MCLFAs) which are produced in sebum are also inhibitory to dermatophyte growth. This may explain the rarity of scalp infections in post-pubertal teenagers and adults as the changes in fatty acid composition of the sebum at this time are thought to result in an increased concentration of MCLFAs. The chief effector cells active against dermatophyte fungi are neutrophils (PMNs) and, to a lesser extent, macrophages. The former are important components of the histological response to hair follicle invasion in man and animals and are prominent in the formation of pustular responses and kerions (see below).

In addition cellular immunity mediated through T helper cells and a TH1 response plays a critical role in resistance to infection. The production of cytokines, such as \( \gamma \)-interferon, activates keratinocytes and promotes the secretion of other factors such as adhesion molecules at the site of infection to guide infiltrating inflammatory cells. While immunological mechanisms provide a potential means of defence, the persistence of infection in many
apparently healthy individuals suggests that they are either ineffective or inoperative in some patients. Antigen derived from *Trichophyton* species containing certain reactive epitopes (probably glycopeptides) interferes with T-cell mediated immunity. It has also been shown that, in many chronic anthropophilic infections at other sites such as the feet and the groin, there is an increased incidence of immediate type hypersensitivity, high IgE levels and the production of IgG₄ and Il-4. This suggests that, in such cases, the TH2 pathway, which is less effective in eliminating fungi, is activated. Dermatophytes, therefore, have the capability of modulating host defence mechanisms.
The epidemiology of tinea capitis

There are three main sources of dermatophytes causing scalp ringworm (Table 1).

Anthropophilic infections which spread from child to child may present as sporadic cases. However these infections are contagious and a single case may be a marker for a small focus of tinea capitis. Spread is thought to be mediated through direct contact or via the airborne dissemination of spores but it is not clear to what extent indirect spread, i.e. through contaminated articles such as combs, brushes, hats or caps or furniture is a factor in the dissemination of organisms.

Infections spread from animals to children (zoophilic infections) usually depend on contact also, although there is heavy environmental contamination in the vicinity of an infected domestic pet such as a cat (carpets, furniture, clothes) or some farm animals such as cattle (sheds, wooden gates and implements). Again the importance of indirect spread is unclear although it may not be possible to elicit a history of direct contact between a child and an animal. Infections spread from soil to humans are rare.

Carriage see Carrier State below

The presence of viable organisms without infection, occurs in children or adults who live with or go to school with other infected children. A carrier is defined as an individual without clinical signs and symptoms from whose scalp organisms can be grown in culture, usually in small quantities [7].

Zoophilic infections

Exposure to an infected animal or to a contaminated environment is a prerequisite for infection [3]. The distribution of infection mirrors the geographic range of the animal. The commonest cause of this infection in the UK is *Microsporum canis* [2]. Its geographic range is, however, worldwide as it is spread from cats or dogs. In many parts of the UK *M. canis* infections are
infrequent but still the commonest forms of tinea capitis in those locations. *Microsporum canis* infections are also seen in children who do not have a history of exposure to cats or dogs. The likely explanation is that they have acquired the infection from a contaminated environment. An alternative is that the infection may rarely spread from child to child. While theoretically possible this is likely to be a very uncommon route of infection.

*Trichophyton verrucosum* is the dermatophyte fungus from cattle and, rarely, other ruminants. The worldwide distribution of this infection is not well documented although it occurs sporadically in many countries including the UK and is more prevalent in young animals and where cattle over-winter indoors. A programme for immunisation of cattle against ringworm in Russia and northern Europe has led to a reduction in the number of animal cases and human infections although the disease has not been eliminated.


**Anthropophilic infections**
The geography of the anthropophilic infections has altered considerably over the past few decades. Before this time endemic areas were well defined and some species were confined to specific regions [7]. Over the past thirty years there has been a greater change following increased travel which has allowed species originally endemic in one area to become established in another [8]. This is not necessarily associated with a great upsurge in the numbers of cases. In the UK sporadic cases of scalp infection caused by dermatophytes not originally found in the country, such as *T. violaceum*, occurred regularly through the 80’s and 90’s [9]. But certain organisms have been associated with a significantly increased incidence of infection in certain communities — *T. tonsurans* is probably the best example of this [10]. In the UK the most prevalent infection now is *T. tonsurans*, particularly in cities [11,12], although
*M. canis* is isolated by a wider range of laboratories. In addition other anthropophilic fungi such as *T. violaceum*, *T. soudanense* and *M. audouinii* are also seen in cities [10]. These infections are commoner in black children — *T. violaceum* in children from the Indian subcontinent or East Africa. However children from all ethnic backgrounds are susceptible. The increase in *T. tonsurans* infections in the UK was first noted in 1992 and since then the numbers have increased by 10–40 fold. In the USA *T. tonsurans* is also the most frequent isolate; it also appears to be commoner in urban populations, particularly black American children, than in other cultural or ethnic groups [13,14]. A summary of the pattern of infection in children from different geographic areas is shown in Table 2.

Little is known about the risk factors for anthropophilic infection. Those cited by different investigations include: overcrowding within households or schools, hairdressing salons, use of shared combs and ethnicity [11,13]. The current spread of *T. tonsurans* in the USA, Europe and South America is most often seen in black communities; this species has also recently been found in West and East Africa. This infection also occurs in children from other ethnic backgrounds. Ethnicity, social and cultural factors, and hair styling may all play a role in determining the spread of infection [15]. However definitive proof is lacking.

**Summary**

It is clear that the pattern of tinea capitis in the UK has changed in the past ten years with a significant rise in the incidence and prevalence of cases of infection due to *Trichophyton tonsurans*. This is reflected by major changes in the level of diagnostic work seen in some centres. For instance in South London the number of cases diagnosed annually at the St Johns Institute of Dermatology has increased by more than 40-fold over this period. The main focus of this epidemic has been cities where there are either long-standing or more recently established black communities. But it is clear that infection can occur in any child irrespective of their ethnic origin. It is therefore important to continue surveillance of this problem, to understand the specific needs of the
ethnic minority communities, who have been the main focus of this infection, and to ensure that methods are in place to control this new outbreak of a common childhood condition.
Clinical detection

The main clinical characteristic of tinea capitis is hair loss which is often accompanied by scaling [2,15] (figure 1). In addition there are symptoms such as itching and, more rarely, pain. Expression of both is highly variable and often infections are asymptomatic. Hair loss may develop in single or multiple patches, but in addition individual hairs or small hair clusters may be involved. Hairs may be broken either above or at scalp level; where swollen broken hair stubs are prominent the pattern is known as black dot ringworm (figure 2). Scaling may occur either in the presence or absence of hair loss and signs of inflammation such as erythema or pustule formation are variably expressed (figure 3). In very inflamed lesions an overlying crust may form. A kerion (kerion Celsi) is a highly inflamed lesion of tinea capitis (figure 4). Lesions occur unpredictably and, although commoner with zoophilic infections, they also occur with anthropophilic ringworm. Kerions are large boggy masses of inflammatory tissue with pustules. If no crust is present pus obtained directly from pustules of a kerion seldom contains bacteria. However *Staph. aureus* may be isolated from beneath crusts and removing the surface debris is an important component of the treatment.

It is difficult to separate the different causes of tinea capitis on clinical grounds alone.

Ectothrix infections, as exemplified by those caused by *M. canis*, are generally inflamed and the broken hairs are often seen projecting above the scalp skin. Hair loss usually occurs in defined patches. Infections due to *T. verrucosum* appear similar but there is a high frequency of pustule formation and kerions occur regularly. By contrast, endothrix infections show a greater variation in clinical appearances [15] and although many cases do not show the characteristics of severe inflammation seen with so many ectothrix infections, scattered pustular reactions as well as kerions can also occur. Other patterns include: hair loss without significant scaling, black dot ringworm, minimal but diffuse hair loss, localised scaling resembling seborrhoeic dermatitis.
(dandruff). In the early stages favus infections, caused by *T. schoenleini*, do not differ clinically from other forms of tinea capitis. As the disease develops lesions become more prominent and a dense white to yellow crust representing the mass of hyphae and keratin debris, the scutulum, around hair follicles, appears together with cicatricial or scarring alopecia [16].

Other clues helpful in the clinical diagnosis of tinea capitis include the presence of tinea corporis at other sites particularly the face or upper trunk. These lesions are typically annular with a raised scaly margin and multiple lesions are common. Onychomycosis due to the organisms that cause endothrix infections is seldom seen in children with tinea capitis but may occur in adults from the same household.

A number of conditions can be confused with tinea capitis. In alopecia areata there is seldom inflammation in the area of alopecia and neither scaling nor itching. Seborrhoeic dermatitis occurs in children at all ages. The scaling is usually diffusely scattered and there is seldom associated hair loss. Other scalp conditions with more scaling include psoriasis and pityriasis amiantacea.

**Summary**

It is unreliable to depend on clinical diagnosis alone to identify cases of tinea capitis given the range of clinical expression and the high numbers of children with mild infections that are difficult to detect. Laboratory methods should be used wherever possible to confirm the diagnosis.
The Carrier State

Children or adults without visible signs of tinea capitis and without evidence, on direct microscopy, of hair shaft invasion, but from whose scalps dermatophytes are grown are termed carriers. Carriage is therefore defined by the absence of clinical or laboratory evidence of hair shaft invasion. However the source of the positive cultures is not clear. It is apparent that in some individuals there is temporary carriage of fungi, for instance amongst laboratory staff after examining and sampling a patient, and it is presumed that in these individuals carriage simply represents temporary presence of arthroconidia within the scalp. Usually growth of fungus from scalp brushes from these individuals is sparse with 1-5 colonies being found per brush sample. Equally surveys conducted in school have identified children with positive cultures and absent signs and long term follow-up has shown that in a proportion of these individuals the cultures become negative. It has also been found, mainly in *T. tonsurans* infections, that careful and detailed examination will reveal that in some children with low colony counts there are scattered infected hairs which have positive microscopy. It requires a long and detailed examination of the scalp to detect these individuals. It is likely, therefore, that although in some individuals there is carriage (without hair invasion) of arthroconidia, others have very limited, but genuine, infections and that these are difficult to separate from true carriers without detailed clinical examination. This presents a difficulty in organising adequate control (see below).

Summary

Carriage of fungi, defined as positive cultures taken by brush sampling but absence of clinical signs of infection or positive direct microscopy of hair, can occur. However in the case of *T. tonsurans* infection in some individuals it is possible to overlook limited and symptom-free infections accompanied by hair shaft invasion without highly detailed examination of the scalp.
Diagnosis

The diagnosis of tinea capitis is based on clinical inspection together with the appropriate use of laboratory diagnostic tests [17]. However, dependence on the clinical diagnosis of tinea capitis is unreliable and has a low specificity [11,16] even though certain signs such as lymphadenopathy are useful predictors of infection [18]. For this reason, wherever possible, the diagnosis should be confirmed by appropriate laboratory tests.

Wood's light

Filtered ultraviolet (Wood’s) light elicits a green fluorescence from some dermatophyte fungi, mainly *Microsporum* species, in hair infections. Exposure to Wood’s light is a useful screening procedure for taking specimens from *Microsporum* infections. Equally it is unhelpful in many of the anthropophilic infections seen in the UK as they do not fluoresce.

Taking scalp samples for laboratory diagnosis

There are a number of different approaches to taking scalp samples. The most complete method involves taking scrapings, containing hairs, which can be used for both direct microscopy and culture and is the best method of obtaining infected hairs or hair remnants, the chief sources. Ideally scrapings should be taken with a blunt solid scalpel [17]. A disposable scalpel blade can be used but should be held vertically to the skin. Lesions of tinea capitis, particularly those that are inflamed are often tender, and children are apt to move suddenly. If the infection is due to an organism which is fluorescent under Wood’s light, it is often possible to use this to select individual hairs which can be pulled from the scalp with a pair of tweezers or forceps without discomfort. It is important to obtain as much material as possible as this will maximise the chances of a positive result. Ideally scraping should focus on areas where there is clinical abnormality and this method is not suitable for detecting carriers. Scalp scrapings provide the best means of making a diagnosis and distinguishing between carriage and infection.
Scalp brushings

Brushes can be used to detect carriers as well as infected patients. Generally the technique relies on the growth of fungi from the prongs of the brush although it is possible to remove hair and skin samples from brushes for direct microscopy. It is not as accurate as skin scraping, though, for detecting infection because it yields less suitable material for microscopy. The original method for obtaining brushings was to use a disposable (sterilizable) plastic scalp brush [19]. A satisfactory alternative is a disposable unpasted toothbrush of the type which can be stored in its own cylindrical container. These brushes are cheap and can be obtained in batches. They may also be discarded after use and can be sent by post with ease. Alternative sampling techniques include moistened swabs or squares made of velvet or other suitable materials. Brushes are passed through the hair firmly, but without undue force, several times. Ideally they should be used in the area where there is clinical abnormality. Brushes may also be used to detect the presence of fungi in suspected carriers where there will be no observable lesions and here the brushes should be passed through different areas of the scalp.

Sending materials to the laboratory

It is important that the material is sent to a laboratory with some experience in mycological isolation techniques. The recognition of infected hairs and the identification of the organisms can be difficult and training is necessary. Scrapings should be sent folded in dark paper or in special sample packs. While they will survive several days or longer it is important to send them to the laboratory as soon as possible. This is particularly important with kerion samples as contaminating bacteria may overgrow any dermatophytes in lesions. Results of direct microscopy should be available within 24 hours of receipt by the laboratory but cultures will take 2-3 weeks longer.

Direct microscopy

Direct microscopy is performed usually by immersion of the samples in 10-20% potassium hydroxide (KOH). Unlike nail samples where the longer the
specimen is kept the better the preparation it is important to examine hair samples treated with KOH within 30 min and without vigorous squashing of the specimen on the slide. The reason is that it is important to see whether arthroconidia lie around or within the hair shaft. If a heavily infected hair with an endothrix infection is exposed to KOH for too long or if the preparation is squashed too vigorously the endothrix spores released from the hair interior may appear to coat the outside of the hair giving rise to the appearance of ectothrix invasion. Treatment advice can be initiated on the basis of a positive direct microscopy before culture results are available. For instance endothrix infections are anthropophilic.

**Culture**
The conventional culture medium used for dermatophyte fungi is Sabouraud (glucose/peptone) agar [2,17]; 4% malt extract agar is an alternative. Antibiotics such as chloramphenicol and cycloheximide are added to the medium to prevent overgrowth of bacteria or non-dermatophyte mould fungi. The appearances of dermatophytes may vary with the composition of the medium and it is important that the same supplier is used for purchase. Petri dishes are excellent for viewing cultured materials and are important if the brush sampling methods have been used. If scrapings are taken Petri dishes or screw capped bottles or tubes may be used. Dermatophytes grow from inoculated hair samples. If brushes are used a heavy growth is indicative of an infection; brush samples from carriers usually yield a scanty growth.

Scrapings with hairs, as well as brushes, are gently pressed into the agar surface, the former with a sterile needle. Most dermatophytes grow at 26° C and plates can be examined after 2 weeks. Details of the identification procedures are beyond the scope of this article.

If unsuitable material is sent the report should indicate that the result is unreliable.
Summary
Wherever possible scalp scrapings, including hairs and hair fragments, should be used as the primary method of detection of cases. However this may be difficult to accomplish in many instances and, therefore, a second line approach would be the use of sterile brushes such as disposable toothbrushes. Cultures should be repeated after therapy. Ideally the annual diagnostic figures for tinea capitis should be collected from a number of sentinel diagnostic laboratories in order to monitor the progress of this epidemic and the effect of control measures.
Management of tinea capitis

The main goals of treatment of tinea capitis are treatment of the patient and prevention of spread to other children [1]. This involves the identification of infected patients, treatment of the patient, checking mycological recovery and identification of human contacts (anthropophilic infections) determining which are infected and which are carriers. If zoophilic infections are suspected animal contacts and other children in the household should be screened for similar infection. Contacts, whether carriers or infected cases, should be treated.

Scalp ringworm was one of the earliest described skin diseases and topical treatments, involving mercurials, tars and, later, dyes were used extensively. There is little evidence that any were effective and children with tinea capitis were often segregated or excluded from school. Adjunctive treatments such as scalp shaving prior to application of topical medications were frequently used. With the wider use of irradiation-based treatments in dermatology X-ray epilation of tinea capitis was commonly employed at the beginning of the twentieth century. The long term consequences of this treatment included radiodermatitis and dysplastic lesions or non-melanoma skin cancer. The discovery of the oral antifungal, griseofulvin, provided the first great breakthrough in the management of the condition. It is now recognised that the best method of treating tinea capitis is through oral medication. Topical agents in current use do not eradicate hair shaft infection and although there may be temporary improvement this is followed by relapse in most cases.

Treatment of cases
Griseofulvin
The longest standing effective treatment for tinea capitis is griseofulvin given in a dose of 10mg/kg daily for a period of 6-8 weeks [20]. Griseofulvin is an orally active compound derived from a Penicillium species [21]. It is fungistatic in vitro and its mode of action is through the inhibition of the formation of
intracellular microtubules. Griseofulvin is active in vitro against dermatophyte fungi but few other organisms respond to the drug.

There are both tablet and liquid formulations of griseofulvin. However, in the UK production of the liquid (paediatric) formulation of griseofulvin has been discontinued by the main supplier. Alternative formulations can be imported or some pharmacies suspend crushed tablets of griseofulvin in a suitable liquid base. However neither approach has been approved by a UK licensing authority. In assessing the evidence for its efficacy it is important to recognise that, because griseofulvin was one of the earliest antifungal drugs introduced, there are few comparative clinical trials. However reported experience suggests that for most organisms causing tinea capitis it is effective, although there are some patients with *M. canis* infections who require longer courses of treatment, e.g. 12 weeks. Since the earliest clinical studies, patients with *T. tonsurans* infections have been reported to have a variable response to griseofulvin and, again, the duration of therapy may have to be increased. The Infectious Disease Working Group from the American Academy of Pediatrics, for instance, recommend a higher dosage, 20mg/kg/day for *T. tonsurans* infections [22]. There are no randomised comparative studies of the two doses (10 v 20 mg/kg day). However there are now some clinical trials comparing terbinafine with griseofulvin [23,24].

There is also evidence that higher intermittent doses of griseofulvin may also be effective against certain endothrix infections, e.g. *T. violaceum* [25]. Regimens vary, but 1000 mg given as a single dose or 500 mg as a stat dose followed by a repeat dose after 2 weeks have been two of the regimens advocated. These regimens were specifically designed for the mass treatment of endemic scalp ringworm. While these high-dose regimens have not been compared with the conventional daily treatments, published results suggest efficacy rates that are slightly lower than with conventional doses and they are not recommended for routine use.
Terbinafine.
Terbinafine is an allylamine drug with broad-spectrum antifungal activity in vitro [26]. It blocks the formation of ergosterol in the fungal cell membrane by inhibiting squalene epoxidase which leads to the accumulation of squalene. It is fungicidal in vitro. It is potent against all dermatophytes causing scalp ringworm in vitro. Terbinafine is available as a cream or in tablet form (250mg). In some countries a paediatric tablet is available (125mg). The dose is 250 mg for adults. In children the treatment regimen used is based on weight: <20kg 62.5 mg/day, 20-40kg 125 mg/day and >40 kg 250 mg/day.

In tinea capitis it is effective against a range of fungi and there are a number of studies comparing terbinafine with griseofulvin [23,24]. The recommended treatment period is usually 4 weeks. Most studies have shown that *T. tonsurans* requires one month of treatment although there is one suggesting that one week may produce similar responses [27]. In a further study of *T. violaceum* infection there was no difference in mycological cure rates at follow up when 1 week of treatment was compared with 2 or 4 weeks [28]. A meta-analysis of studies comparing terbinafine with griseofulvin showed that terbinafine was as effective at treatment durations of up to 2 to 4 weeks for *Trichophyton* infections compared with griseofulvin for 6 to 8 weeks [29]. However the responses of *Microsporum* species are generally slower than those of *Trichophyton* and in some patients there is treatment failure [23,30,31]. However higher doses of terbinafine, more than 6 mg/kg per day, appear to produce good responses [31]. This applies to most *Microsporum* species tested, although treatment failures have mainly been reported with *M. canis*. The reasons for this are not clear although there is little evidence to suggest that *Microsporum* species are significantly less sensitive to terbinafine in vitro than *Trichophyton* [32]. There are also no published clinical trials comparing griseofulvin with terbinafine that include adequate numbers of patients with *Microsporum* infections.

In some countries, but not the UK, a paediatric formulation of the terbinafine tablet (125 mg compared to the adult tablet of 250 mg) is available. For
smaller children it is necessary to break the tablets, which may be scored (depending on source).

Itraconazole

Itraconazole is an orally active triazole antifungal. Its mode of action, as with all azoles, is through the inhibition of the formation of ergosterol in the fungal cell membrane via inhibition of the 14 α-demethylase enzyme. Itraconazole is fungistatic in vitro and is active against a wide range of organism including all dermatophytes. Itraconazole comes in two main formulations: a capsule containing pelleted itraconazole and an oral solution containing itraconazole in cyclodextrin. The latter is designed for the treatment of severe oropharyngeal and oesophageal candidosis in severely immunocompromised patients including children. In the UK it is not approved for paediatric use in dermatophytosis at present.

Itraconazole is also effective in dermatophyte infections. For tinea capitis itraconazole is generally given as 3–5 mg/kg daily doses [33,34,35]. The regimens used have varied between 3–5 mg/kg daily for four to six weeks which is effective in over 80% of children with tinea capitis due to \textit{T. tonsurans}. Other studies have shown a lower level of efficacy eg 40% in one study. There is no evidence to date that, in comparison with griseofulvin, \textit{Microsporum} and \textit{Trichophyton} species differ in their responses to itraconazole. A pulsed regimen using 5 mg/kg for one week every three weeks has been evaluated in a small number of children [36]. Preliminary data suggest that 2-3 pulses will be required for most infections.

The pelleted capsule formulation is difficult to use in children on a dose per weight basis as it involves opening and dividing the contents of capsules.

Fluconazole

Fluconazole is an orally active triazole antifungal. As with other triazoles the main site of action is through the inhibition of cytochrome P450 via the 14-α demethylase enzyme. There are capsule and liquid formulations of fluconazole. The drug is active against a range of fungi including
dermatophytes. Its value in tinea capitis is still the subject of evaluation. The
doses that have been used have ranged from 1.5 to 6 mg/kg daily and up to 8
mg/kg weekly [37,38,39]. The evidence to date suggests that fluconazole is
effective against a range of different organisms including both Trichophyton
and Microsporum species. It also appears to be as effective as griseofulvin
[40].

Topical antifungals
Very little is known about the penetration of topically applied agents applied to
hair. But it is unlikely that any can achieve levels sufficient to provide
sustained growth inhibition or fungicidal activity. One study comparing
miconazole with Whitfield's ointment showed that patients receiving topical
treatment showed some improvement including negative cultures during
therapy [41], but not as great as that expected with oral antifungals. There
was no follow-up in this study. There is also no comparative trial with an oral
agent and topical antifungals do not appear to have a role in the primary
treatment of tinea capitis.

However they may be used as an adjunct to oral therapy to reduce the
frequency of positive cultures during the early stages of therapy. Trials have
been carried out with selenium sulphide [42,43] and ketoconazole shampoo
[44].

Treatment of carriers
In addition to miconazole, mentioned above, topically applied ketoconazole
and selenium sulphide in shampoos reduce the frequency of positive cultures
and, it is argued, they can be used to prevent carriage of organisms in
individuals who, after careful inspection, have no clinical lesions. Topical
antifungals are recommended for carriers: asymptomatic children with positive
brush cultures. If scalp brushes produce very heavy growth of fungus it is
likely that the children have a true but asymptomatic infection and these
should be treated as infected.
Schools
Theoretically, infected children pose a potential risk to non-infected children, although the method by which the organisms spread from head to head is not known. On the other hand the infected child is likely to have been at school for some time before detection of the infection. Exclusion from school merely reinforces the child’s isolation and is probably too late to prevent spread.

Treatment of kerions
In kerions the same treatment strategy as that given for normal infections is used. It is more difficult, though, to ensure that lesions are sterile after 6-8 weeks and often it is necessary to continue therapy for longer e.g. 12-16 weeks. In addition there is some uncertainty over the need for anti-inflammatory treatments. For instance there have been few clinical trials which have addressed the issue of the use of systemic corticosteroids in kerions and advice is usually based on anecdotal experience. One such trial which examined the value of such treatment found that corticosteroids made no difference to clinical and mycological response rates [45].

Removal of surface crusts is often helpful as it relieves itching and secondary infection. It can be painful and therefore the procedure should be carried out gently after soaking with lukewarm water or saline applied as topically in moistened dressings. The softened crusts can then be gently teased away. Sometimes secondary bacterial infection, usually due to Staph. aureus, should be treated with antibiotics such as flucloxacillin and the application of an antifungal cream which also has anti-Gram-positive activity (miconazole, clotrimazole, econazole) may allow the scalp to heal and prevent formation of new crusts.

Summary
For the time being it is important to recognise that there is no approved treatment for tinea capitis in childhood in the UK apart from the tablet formulation of griseofulvin. There are a number of options. Children with tinea capitis infections are treated with griseofulvin at a dose of at least
10 mg/kg; this may be increased to 20 mg/kg in patients with
*T. tonsurans* infection or where there is failure to respond after 6 weeks
of treatment. Although there is no UK approved liquid paediatric
formulation of griseofulvin, it is still an effective treatment with evidence
that it provides broad cover for all the different organisms that cause
tinea capitis. In younger children its use may involve giving crushed
tablets or suspensions of crushed tablets.
Terbinafine is now well documented as a treatment for *Trichophyton*
infections, particularly those caused by *T. tonsurans*; the duration of
treatment is 4 weeks. It is equivalent to griseofulvin given for 8 weeks
and it is increasingly recommended as the first treatment for *T.
tonsurans* infections. Its dose is doubled in *Microsporum* infections.
Itraconazole and fluconazole are alternatives, particularly in
*Microsporum* infections.

The use of a topical treatment, either selenium sulphide or ketoconazole
shampoo, or another topically active antifungal e.g. terbinafine cream is
recommended at least twice weekly during the first two weeks of therapy
and that children on treatment should not be kept off school unless their
clinical condition warrants it, e.g. severe kerion. Carriers are also given
a topical preparation such as selenium sulphide shampoo at least twice
weekly but do not receive oral antifungals. However if there is heavy
growth of dermatophytes from scalp brushes taken from children with
clinically normal scalps they should be treated as if they are infected
cases i.e with oral therapy. Children in contact with tinea capitis should
be examined very carefully for signs of infection which may be confined
to a few broken hairs. If infected hairs are seen and this is confirmed by
mycological examination the children should receive oral therapy.

The use of systemic corticosteroids for routine treatment of kerions is
not recommended but they may be used at the discretion of the treating
physician in the presence of antifungal therapy if there is a severe
allergic response (dermatophyte id reaction).
Treatment of scalp ringworm can be carried out in primary care and for most cases it is not necessary to refer children to a dermatologist. However the importance of confirming the diagnosis by laboratory procedures, including culture, before starting treatment should be emphasised.
Management of spread

In zoophilic infections it is important to identify a potential source of infection from the case history and, in the case of domestic pets, take the animal for treatment of suspected ringworm.

In anthropophilic infections the initial consultation with the index case provides an opportunity to ask about other children in the household or infections (skin, nail) in adults. Where possible other children in the household should be examined or scalp brush samples taken. Contacts who are carriers should be treated with selenium sulphide or ketoconazole shampoo whereas infected contacts require oral antifungal therapy. Adults in contact with infected children may develop tinea corporis, onychomycosis and, rarely, tinea capitis as well.

Infections in schools can cause considerable anxiety amongst both parents and teachers. The ethical implications of instituting school-wide policies have to be considered carefully. It is important that children, parents and staff are informed about the infection and are reassured that ringworm is completely treatable and not a sign of neglect. Recommendations are provided as an example of a potential way of managing cases. It is still not clear though whether clusters of infection are more likely to be related to spread in the school or the home environment. In areas where cases have occurred information about scalp ringworm should be provided to parents and staff. If more than two children in a class are infected the rest should be screened by scalp brushing after parental consent. Thereafter carriers and infected children should be treated using the guidelines described above and it is important to review classes which have been examined to ensure that new infections do not occur. Screening large numbers of children by clinical examination is not easy. Scalp lesions are often difficult to detect because many children have minimal scaling or hair loss. The same is true if their hair is long or the hairstyle involves complex braiding. For this reason the use of scalp brushes is advised in addition to inspection.
Summary
There has been a change in the pattern of scalp ringworm or tinea capitis in the UK with an increased frequency of infection due to anthropophilic dermatophytes such as *T. tonsurans* that spread from child to child in the community. While the majority of cases occur in urban areas and in black children this infection can affect any child. Treatment measures depend on the use of oral antifungal therapy with griseofulvin. It is important to note, though, that the current measures for control have not been effective where spread of *T. tonsurans* has occurred elsewhere, eg in the USA, possibly due to the difficulty of distinguishing between carriers and children with minimal infections. *T. tonsurans* is not a new infection in the UK and in the 1970’s there were outbreaks of infections in schools [46]. However control was achieved by rigorous surveillance. Although there are some differences in the new pattern of infection improving early detection rates is likely to provide some of the answers.

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### Table 1: Causes of tinea capitis

<table>
<thead>
<tr>
<th>Anthropophilic</th>
<th>Zoophilic</th>
<th>Geophilic</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Ectothrix</strong></td>
<td><strong>Ectothrix</strong></td>
<td><strong>Ectothrix</strong></td>
</tr>
<tr>
<td><em>Microsporum audouinii</em></td>
<td><em>Microsporum canis</em></td>
<td><em>Microsporum gypseum</em></td>
</tr>
<tr>
<td>Includes variants <em>langeronii</em> &amp; <em>rivalieri</em></td>
<td><em>Trichophyton mentagrophytes</em></td>
<td></td>
</tr>
<tr>
<td><em>Microsporum ferrugineum</em></td>
<td><em>Trichophyton verrucosum</em></td>
<td></td>
</tr>
<tr>
<td><strong>Endothrix</strong></td>
<td><strong>Source:</strong> animals, animal fomites <strong>Distribution:</strong> sporadic. Rare outbreaks through exposure to common source. Indirect spread possible</td>
<td><strong>Source:</strong> soil <strong>Distribution:</strong> sporadic</td>
</tr>
<tr>
<td><em>Trichophyton tonsurans</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Trichophyton violaceum</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Trichophyton soudanense</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Trichophyton gourvilli</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Trichophyton yaoundei</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Trichophyton schoenleinii</em></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Source:** children (adults) **Distribution:** in endemic and epidemic foci. Infection in adults seldom involves the scalp but may affect skin or nail

Rarely other *Trichophyton* or *Microsporum* species may cause tinea capitis e.g. *T.rubrum, M. persicolor*

* *Trichophyton schoenleinii* causes favus, which is a clinically distinct form of tinea capitis.*

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Table 2: Tinea capitis – geographic range

<table>
<thead>
<tr>
<th>Area</th>
<th>Dermatophyte</th>
</tr>
</thead>
</table>
| Europe                    | *M. canis*, (*T. verrucosum* – less common)\n*T. tonsurans*, *T. soudanense*, *M. audouini* – including *M. langeroni*
| USA, Canada               | *T. tonsurans*, *M. canis*                                                   |
| Mexico, Central America    | *T. tonsurans*, (*M. canis*)                                                 |
| South America             | *M. canis*                                                                   |
| West Africa               | *M. audouini*, *M. langeroni*, *M. rivalieri*\n*T. soudanense*, *T. yaoundei*, *T. gourvili*                                          |
| East Africa               | *T. violaceum* (north), *T. schoenleinii*\n*M. canis*, *M. audouini*                                                       |
| Middle East               | *M. canis*, *T. violaceum*                                                   |
| Indian subcontinent       | *T. violaceum*, *T. tonsurans*                                               |
| SE Asia                   | *M. canis*, *M. ferrugineum*, *T. tonsurans*,                                |
| Russia and Central Asia   | *M. canis*, *T. violaceum*, *M. ferrugineum*                                 |
| China, Japan, East Asia   | *M. canis*, *T. tonsurans*                                                   |
| Australasia               | *M. canis*                                                                   |
### Table 3: Antifungal drugs used in tinea capitis

<table>
<thead>
<tr>
<th>Antifungal agent</th>
<th>Daily dosage (weekly or intermittent dosage)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Griseofulvin</td>
<td>10 mg/kg/day (some physicians use 20 mg/kg/day for <em>T. tonsurans</em>)</td>
</tr>
<tr>
<td>Terbinafine&lt;sup&gt;1&lt;/sup&gt;</td>
<td>&lt;10 kg 62.5 mg, 10-20 kg 125 mg, &gt;20 kg 250 mg – all daily</td>
</tr>
<tr>
<td>Itraconazole&lt;sup&gt;1&lt;/sup&gt;</td>
<td>2-4 mg/kg/day&lt;sup&gt;2&lt;/sup&gt;. Some data suggest that 5 mg/kg in weekly pulses each month is effective — 2-3 pulses</td>
</tr>
<tr>
<td>Fluconazole&lt;sup&gt;1&lt;/sup&gt;</td>
<td>2-5 mg/kg/day&lt;sup&gt;2&lt;/sup&gt;. Weekly treatment with 8 mg/kg may be as effective</td>
</tr>
</tbody>
</table>

1 - No paediatric licence for this indication at present

2 - These are based on non-comparative trial data

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Figure 1: Tinea capitis due to *T. tonsurans*. The pattern of infection is diffuse with minimal scaling.

Figure 2: Tinea capitis – “black dot ringworm” due to *T. violaceum*. This can be diagnosed in the laboratory by extracting and examining one of the swollen hair stubs.
Figure 3: Tinea capitis – *Microsporum canis*. The appearance is inflammatory with dense scaling and erythema

Figure 4: Inflammatory tinea capitis due to *T. tonsurans*. This child has a kerion with pustules. Examination is difficult as the scalp is very tender
References


Useful sources of information

British Association of Dermatologists
http://www.bad.org.uk

Health Protection Agency
Includes topics on Fungal infection, Schools factsheets and Schools guidance
http://www.hpa.org.uk

National electronic Library of Infection (NeLI)
http://www.neli.org.uk

NHS Direct
http://www.nhsdirect.nhs.uk

NHS Prodigy Guidance
http://cks.library.nhs.uk

Wired for Health
Health information for a range of audiences that relates to the National Curriculum and the National Healthy Schools Programme
http://www.wiredforhealth.gov.uk