I'm not a bot

Hair loss can be caused by many factors, including nutrient deficiencies, stress, certain health conditions, and changes in hormone levels. Early diagnosis and treatment may help stop hair loss and promote hair regrowth.But it can be more severe.In people assigned female at birth (AFAB), noticeable hair loss is pretty common, with around one third experiencing it at some point. However, whether its subtle thinning all over or a bare patch where the scalp can be seen, it can look different to the typical baldness you might expect.From dietary deficiencies to stress, hair loss in AFAB folks can have a number of causes.Telogen effluvium when significantly more hairs move from the growing to the shedding stage can occur after a traumatic or stressful experience, such as: childbirthextreme weight loss,the loss of a loved one Triggers for other types of hair loss range from inflammatory scalp conditions, like eczema, to an underlying health concern, such as an autoimmune condition. Even tight hairstyles, like ponytails or braids, can lead to hair loss as a result of putting pressure on roots. Female pattern baldness also known as androgenetic alopecia is hair loss that affects people assigned female at birth.Female pattern baldness is usually hereditary caused by a genetically shorter hair-growing period and a longer period between the shedding and growth phases. Genes from parents may also affect the hair, causing smaller follicles and thinner strands. However, age and hormones may play a part, too, as its more common after menopause when estrogen levels reduce.This means that the effects of male androgen hormones which are linked to male pattern baldness may be greater.More androgenetic activity can also occur through an underlying endocrine condition, such as a tumor on the ovary gland. In female pattern baldness, the hairs growing phase slows down. It also takes longer for new hair to begin growing. Hair follicles shrink, leading the hair that does grow to be thinner and finer. This can result in hair that easily breaks.People with this condition also tend to shed more hairs than the average person, though complete baldness is less likely.In male pattern baldness, hair loss starts in the front of the head and recedes to the back until the person goes bald.But female pattern baldness starts at the part line, sometimes appearing all over the head. Hair at the temples may also recede.Doctors divide the condition into three types:Type I is a small amount of thinning that starts around the part.Type II involves widening of the part and increased thinning around it.Type III is thinning throughout, with a see-through area at the top of the scalp.If you have female pattern baldness, you may be able to camouflage the hair loss at first by adopting a new hairstyle.But it often becomes too difficult to hide the thinning hair.Early diagnosis is encouraged, as it can enable you to start a treatment plan and potentially minimize future hair loss. Your treatment plan will likely consist of one or more medications approved to treat the condition.Minoxidil (Rogaine) is the only drug approved by the Food and Drug Administration (FDA) to treat female pattern baldness.Its available in 2% or 5% formulas. If possible, opt for the 5% formula older studies found that its superior. Apply minoxidil to your scalp every day. Though it wont fully restore the hair youve lost, it can grow back a significant amount of hair and give an overall thicker appearance. It can take around 6 to 12 months to see results. And youll need to keep using minoxidil to maintain the effect, or itll stop working. If this happens, your hair may return to its previous appearance.The following side effects are possible:rednessdrynessitchinghair growth on areas where you didnt want it, such as your cheeksFinasteride (Propecia) and dutasteride (Avodart) are FDA approved to treat male pattern hair loss. Theyre not approved for female pattern hair loss, but some doctors do recommend them.Studies are mixed about the effectiveness of these drugs for AFAB folks, but some research shows that they do help regrow hair in female pattern baldness. Side effects can include: headacheshot flashesdecreased sex drive, especially during the first year of usePeople also should avoid becoming pregnant, because it can increase the risk for birth defects.Spironolactone (Aldactone) is a diuretic, which means it removes excess fluid from the body. It also blocks androgen production and therefore may help regrow hair thats been lost as a result of female pattern baldness. This medication can cause a number of side effects, including:electrolyte imbalancesfatiguespotting between periodsirregular menstruationtender breastsYou may need to have regular blood pressure and electrolyte tests while you take it. If youre pregnant or plan to become pregnant, you shouldnt use this medication due to the risk of birth defects. Laser combs and helmets are also FDA approved to treat hair loss. They use light energy to stimulate hair regrowth, but more research needs to be done to determine if this is truly effective.Platelet-rich plasma therapy may also be beneficial. This involves drawing your blood, spinning it down, then injecting your own platelets back into your scalp to stimulate hair growth. Though promising, more studies need to be done.Similarly, there isnt any evidence that taking iron will regrow your hair. But if low iron is contributing to your hair loss, a doctor or other healthcare professional still might prescribe an iron supplement. Other supplements, such as biotin and folic acid, are also promoted to thicken hair. A 2015 study showed that people developed thicker hair after taking omega-3 fatty acids, omega-6 fatty acids, and antioxidants. However, its best to check with a healthcare professional before taking any supplements with this aim.If you want a simple way to conceal hair loss, you might try a wig or spray hair product.A hair transplant is a more permanent solution. During this procedure, a healthcare professional removes a thin strip of hair from one part of your scalp and implants it in an area where youre missing hair. The graft regrows like your natural hair.A doctor or dermatologist can give a diagnosis for thinning hair. Testing generally isnt necessary, but theyll examine your scalp to see the pattern of hair loss. If they suspect another type of hair loss other than female pattern baldness, they may also perform a blood test to check your levels of thyroid hormone, androgens, iron, or other substances that can affect hair growth. Hair loss is passed down from biological parents to their children, and many genes are involved. You can inherit these genes from either biological parent.Youre more likely to develop female pattern baldness if your biological parents or other close genetic relatives have experienced hair loss. Female pattern baldness is generally caused by an underlying endocrine condition or a hormone-secreting tumor. You could consult a healthcare professional if you have other symptoms, such as: irregular periodsevere acneincrease in unwanted hairThese may be a sign that youre experiencing a different type of hair loss.People are less likely to develop female pattern baldness before midlife and are more likely to start losing hair once they get into their 40s, 50s, and beyond. While some forms of AFAB hair loss are temporary, female pattern baldness is permanent and irreversible without treatment.However, proper treatment can stop the hair loss and potentially help regrow some lost hair. Youll need to stay on this treatment long-term to prevent losing your hair again. Female pattern baldness will progress without treatment. However, progression is often slow, taking years to even decades to worsen.You might notice periods of stability followed by more rapid hair loss phases. And the earlier you experience female pattern baldness, the quicker it may progress.You cant prevent it, but you can protect your hair from breakage and loss via the following:If youre noticing hair loss, consider reaching out to a doctor or dermatologist. Theyll be able to figure out what kind of hair loss it is and what could potentially be causing it.Plus, theyll be able to recommend and prescribe the best form of treatment. The sooner you receive treatment, the faster youll be able to stop the loss and possibly even regrow some of your hair.Healthline has strict sourcing guidelines and relies on peer-reviewed studies, academic research institutions, and medical journals and associations. We only use quality, credible sources to ensure content accuracy and integrity. You can learn more about how we ensure our content is accurate and current by reading our editorial policy. Wimpole Clinic Limited, incorporated in England & Wales (registration number 08072544), with its registered office at 2 Harley Street, West Central London, London, W1G 9PA. Wimpole Clinic Limited is authorised and regulated by the Financial Conduct Authority and entered on the Financial Services Register, reference number 698329. Female pattern baldness is a type of hair loss that affects women. It causes you to lose hair on the skin covering your head (scalp). Your hair doesnt grow back without treatment.You may hear your healthcare provider refer to female pattern baldness as female pattern hair loss or androgenic alopecia. The medical term for male pattern baldness is also androgenic alopecia. Male pattern baldness is similar to female pattern baldness but it affects men.What are the stages of female pattern baldness?There are five stages of female pattern baldness according to the Sinclair scale:Stage 1: Little or no hair loss.Stage 2: A slight gap appears in your center hair part.Stage 3: A wider gap is present in your center hair part, and theres hair loss on either side of your part line.Stage 4: Bald spots appear toward the front of your hairline.Stage 5: Advanced hair loss.Can you reverse female pattern baldness?If youve lost hair from female pattern baldness, it wont grow back without treatment. Proper treatment can stop hair loss and help regrow some hair.How common is female pattern baldness?Female pattern baldness is common. Its the most significant cause of hair loss in women. Approximately 30 million women in the United States have female pattern baldness.How does female pattern baldness affect my body?Female pattern baldness causes the small, tube-like structures in your scalp that grow your hair (hair follicles) to shrink gradually. As your hair follicles shrink, your individual hairs get thinner and shorter. Over time, those hairs stop growing.Female pattern baldness doesnt affect your physical health. But it can affect you psychosocially (how society and social groups affect your thoughts and emotions). It can also affect you psychologically (how you think about yourself and your behavior). You may experience emotional stress, anxiety and depression.,the free encyclopedia that anyone can edit.117,185 active editors 7,001,826 articles in EnglishThe English-language Wikipedia thanks its contributors for creating more than seven million articles! Learn how you can take part in the encyclopedia's continued improvement.Members of the victorious Blondie crewThe Boat Race 2018 took place on 24March. Held annually, The Boat Race is a side-by-side rowing race between crews from the universities of Oxford and Cambridge along a 4.2-mile (6.8km) tidal stretch of the River Thames in south-west London, England. For the third time in the history of the event, the men's, the women's and both reserves' races were all held on the Tideway on the same day. The women's race saw Cambridge lead from the start, eventually winning by a considerable margin to take the overall record to 4330 in their favour. In the women's reserve race, Cambridge's Blondie (crew pictured) defeated Oxford's Osiris by nine lengths. The men's reserve race was won by Cambridge's Goldie, who defeated Oxford's Isis by a margin of four lengths. The men's race was the final event of the day and completed a whitewash as Cambridge won, taking the overall record to 8380 in their favour. The races were watched by around 250,000 spectators live, and broadcast around the world. (Fullarticle...)Recently featured: Radar, Gun Laying, Mk.I and Mk.IIAndrea NavageroNosy KombaArchiveBy emailMore featured articlesAboutKitty Marion... that Kitty Marion (pictured) was force-fed over 200 times during a hunger strike?... that the North Korean destroyer Choe Hyon is the largest ship constructed for the Korean People's Navy?... that after the release of High and Low, director Akira Kurosawa received telephone calls imitating his film that threatened to kidnap his daughter?... that May Bradford Shockley is why Silicon Valley is where it is?... that the conservation of a goat might endanger the survival of Aquilegia paui?... that Joy Laking predicted in a school writing assignment that within ten years she would be making a living as an artist?... that the Taiwanese restaurant chain Formosa Chang drew inspiration from McDonald's for its non-greasy atmosphere and corporate practices?... that Haridas Mitra had his death sentence commuted after the intervention of Mahatma Gandhi?... that "Steve's Lava Chicken" recently became the shortest song to enter the UK Top 40?ArchiveStart a new articleNominate an articleNgg wa Thiong'oKenyan writer and activist Ngg wa Thiong'o (pictured) dies at the age of 87.In sumo, nosato Daiki is promoted to yokozuna.In association football, Liverpool win the Premier League title.In motor racing, lex Palou wins the Indianapolis 500.In basketball, the EuroLeague concludes with Fenerbahe winning the Final Four Playoff.Ongoing: Gaza warM23 campaignRussian invasion of UkrainetimelineSudanese civil wartimelineRecent deaths: Harrison Ruffin TylerPhil RobertsonMary K. GaillardPeter DavidAlan YentobGerry ConnollyNominate an articleMay 31: Dragon Boat Festival in China and Taiwan (2025); World No Tobacco DayBessarion455 Petronius Maximus, the ruler of the Western Roman Empire, was stoned to death by a mob as he fled Rome ahead of the arrival of a Vandal force that sacked the city.1223 Mongol invasion of Kievan Rus': Mongol forces defeated a Kievan Rus' army at the Battle of the Kalka River in present-day Ukraine.1468 Cardinal Bessarion (pictured) announced his donation of 746 Greek and Latin codices to the Republic of Venice, forming the Biblioteca Marciana.1935 A magnitude-7.7 earthquake struck Balochistan in British India, now part of Pakistan, killing between 30,000 and 60,000 people.2013 A tornado struck Central Oklahoma, killing eight people and injuring more than 150 others.Albertino Mussato (d.1329)Joseph Grimaldi (d.1837)Dina Boluarte (b.1962)Mbaye Diagne (d.1994)More anniversaries: May 30May 31June 1ArchiveBy emailList of days of the yearAboutCucumis metuliferus, the African horned cucumber, is an annual vine in the cucumber and melon family, Cucurbitaceae. Its fruit has horn-like spines, hence the name "horned melon". The ripe fruit has orange skin and lime-green, jelly-like flesh. It is native to Southern Africa, where it is a traditional food. Along with the gemsbok cucumber and the citron melon, it is one of the few sources of water during the dry season in the Kalahari Desert. This photograph, which was focus-stacked from 25 separate images, shows two C.metuliferus fruits, one whole and the other in cross-section.Photograph credit: Ivar LeidusRecently featured: Ignace TonenAustralian white ibisHell Gate BridgeArchiveMore featured picturesCommunity portal The central hub for editors, with resources, links, tasks, and announcements.Village pump Forum for discussions about Wikipedia itself, including policies and technical issues.Site news Sources of news about Wikipedia and the broader Wikimedia movement.Teahouse Ask basic questions about using or editing Wikipedia.Help desk Ask questions about using or editing Wikipedia.Reference desk Ask research questions about encyclopedic topics.Content portals A unique way to navigate the encyclopedia.Wikipedia is written by volunteer editors and hosted by the Wikimedia Foundation, a non-profit organization that also hosts a range of other volunteer projects: CommonsFree media repository MediaWikiWiki software development Meta-WikiWikimedia project coordination WikibooksFree textbooks and manuals WikidataFree knowledge base WikinewsFree-content news WikiquoteCollection of quotations WikisourceFree-content library WikispeciesDirectory of species WikiversityFree learning tools WikivoyageFree travel guide WiktionaryDictionary and thesaurusThis Wikipedia is written in English. Many other Wikipedias are available; some of the largest are listed below. 1,000,000+ articles DeutschEspaolFranaisItalianoNederlandsPolskiPortugusSvenskaTing Vit 250,000+ articles Bahasa IndonesiaBahasa MelayuBn-lm-gCatalетinaDanskEestiEsperantoEuskaraMagyarNorsk bokmlRomnSimple EnglishSloveninaSrpskiSrpskohrvatskiSuomiTrkeOzbekcha 50,000+ articles AsturianuAzrbaycancaBosanskiFryskGaeilgeGalegoHrvatskiKurdLatvieuLietuviNorsk nynorskShqipSlovenina Retrieved from " 2This article is about the year 455. For other uses, see 455 (disambiguation).This article needs additional citations for verification. Please help improve this article by adding citations to reliable sources. Unsourced material may be challenged and removed.Find sources:"455"news newspapers books scholar JSTOR (April 2019) (Learn how and when to remove this message)Calendar yearYearsMillennium1stmillenniumCenturies4thcentury5thcentury 6thcenturyDecades430s440s450s 460s470sYears452453454455 456457458vte455 by topicLeadersPolitical entitiesState leadersReligious leadersCategoriesBirthsDeathsDisestablishmentsvte455 in various calendarsGregorian calendar455CDLVAb urbe condita1208Assyrian calendar5205Balinese saka calendar376377Bengali calendar139 138Berber calendar1405Buddhist calendar999Burmese calendar183Byzantine calendar59635964Chinese calendar (WoodHorse)3152 or 2945to(WoodGoat)3153 or 2946Coptic calendar171172Discordian calendar1621Ethiopian calendar447448Hebrew calendar42154216Hindu calendars- Vikram Samvat511512- Shaka Samvat376377- Kali Yuga35553556Holocene calendar10455Iranian calendar167 BP 166 BPIslamic calendar172 BH 171 BHJavanese calendar340341Julian calendar455CDLVKorean calendar2788Minguo calendar1457 before ROC1457Nanakshahi calendar1013Seleucid era766/767 AGThai solar calendar997998Tibetan calendar(male Wood-Horse)581 or 200 or 572to(female Wood-Goat)582 or 201 or 571King Genseric sacks Rome (455)Year 455 (CDLV) was a common year starting on Saturday of the Julian calendar. At the time, it was known as the Year of the Consulship of Valentinianus and Anthemius (or, less frequently, year 1208 Ab urbe condita). The denomination 455 for this year has been used since the early medieval period, when the Anno Domini calendar era became the prevalent method in Europe for naming years.March 16 Emperor Valentinian III, age 35, is assassinated by two Hunnic retainers of the late Flavius Aetius, while training with the bow on the Campus Martius (Rome), ending the Theodosian dynasty. His primicerius sacri cubiculi, Heraclius, is also murdered.March 17 Petronius Maximus, former domesticus ("elite bodyguard") of Aetius, becomes (with support of the Roman Senate) emperor of the Western Roman Empire. He secures the throne by bribing officials of the imperial palace. Maximus consolidates his power by a forced marriage with Licinia Eudoxia, widow of Valentinian III.Maximus appoints Avitus, most trusted general, to the rank of magister militum and sends him on an embassy to Toulouse, to gain the support of the Visigoths. He elevates his son Palladius to Caesar and has him marry Eudocia, eldest daughter of Valentinian III.May 31 Maximus is stoned to death by an angry mob while fleeing Rome. A widespread panic occurs when many citizens hear the news that the Vandals are plundering the Italian mainland.June 2 Sack of Rome: King Genseric leads the Vandals into Rome, after he has promised Pope Leo I not to burn and plunder the city. Genseric sacks the city for a period of two weeks. Eudoxia and her daughters, Eudocia and Placidia, are taken hostage. The loot is sent to the harbour of Ostia and loaded into ships, from whence the Vandals depart and return to Carthage.July 9 Avitus is proclaimed Roman emperor at Toulouse, and later recognised by the Gallic chiefs in Viernum (near Arles).September 21 Avitus enters Rome with a Gallic army. He restores the imperial authority in Noricum (modern Austria) and leaves a Gothic force under Remistus, Visigoth general (magister militum), at Ravenna.The Ostrogoths conquer Pannonia and Dalmatia.Battle of Aylesford: Prince Vortimer rebels against the pro-Anglo-Saxon policies of his father, Vortigern. He is defeated in the battle at Aylesford (Kent). Hengist and his son Oisc become king of Kent. Horsa and Catigern, brother of Vortimer, are killed. The Britons withdraw to London (according to the Anglo-Saxon Chronicle).Skandagupta succeeds Kumaragupta I as ruler of the Gupta Empire (India). During his reign he crushes the Hun invasion; however, the expense of the wars drains the empire's resources and contributes to its decline.Gaero becomes king of the Korean kingdom of Baekje.[1]Earliest recorded date at Chichen Itza on the Yucatn Peninsula (Mexico) (approximate date).Barter economy replaces organized trade as Romans and other citizens desert their towns for the countryside, where they will be less vulnerable to barbarian raids (approximate date).The city of Vindobona (Vienna) is struck by an epidemic that spreads through the Roman provinces. The disease is probably streptococcus or a form of scarlet fever with streptococcus pneumoniae (approximate date).Rusticus, archbishop of Lyon (approximate date)Wang Baoming, empress of the Southern Qi (d. 512)March 16Valentinian III, emperor of the Western Roman Empire (b. 419)Heraclius, Roman courtier (primicerius sacri cubiculi)May 31 Petronius Maximus, emperor of the Western Roman EmpireBiyu of Baekje, king of Baekje[1]Catigern, prince and son of Vortigern (approximate date)Horsa, leader of the Anglo-Saxons (approximate date)Kumaragupta I, ruler of the Gupta Empire (India)Niall Noigiallach, High King of Ireland (approximate date)Palladius, son of Petronius Maximus (approximate date)Prosper of Aquitaine, disciple and Christian writer (approximate date)^ a b "List of Rulers of Korea". www.metmuseum.org. Retrieved April 20, 2019.Retrieved from " 3One hundred years, from 301 to 400Millennia1stmillenniumCenturies3rdcentury4thcentury5thcenturyTimelines3rdcentury4thcentury5thcenturyState leaders3rdcentury4thcentury5thcenturyDecades300s310s320s330s340s350s360s370s380s390sCategories:Births Deaths Establishments DisestablishmentsvteEastern Hemisphere at the beginning of the 4th century CE.Eastern Hemisphere at the end of the 4th century CE.The 4th century was the time period from 301 CE (represented by the Roman numerals CCCI) to 400 CE (CD) in accordance with the Julian calendar. In the West, the early part of the century was shaped by Constantine the Great, who became the first Roman emperor to adopt Christianity. Gaining sole reign of the empire, he is also noted for re-establishing a single imperial capital, choosing the site of ancient Byzantium in 330 (over the current capitals, which had effectively been changed by Diocletian's reforms to Milan in the West, and Nicomedeia in the East) to build the city soon called Nova Roma (New Rome); it was later renamed Constantinople in his honor.The last emperor to control both the eastern and western halves of the empire was Theodosius I. As the century progressed after his death, it became increasingly apparent that the empire had changed in many ways since the time of Augustus. The two-emperor system originally established by Diocletian in the previous century fell into regular practice, and the east continued to grow in importance as a centre of trade and imperial power, while Rome itself diminished greatly in importance due to its location far from potential trouble spots, like Central Europe and the East. Late in the century Christianity became the official state religion, and the empire's old pagan culture began to disappear.[citation needed] General prosperity was felt throughout this period, but recurring invasions by Germanic tribes plagued the empire from 376[1][2] CE onward. These early invasions marked the beginning of the end for the Western Roman Empire.In China, the Jin dynasty, which had united the nation prior in 280, began rapidly facing trouble by the start of the century due to political infighting, which led to the insurrections of the northern barbarian tribes (starting the Sixteen Kingdoms period), which quickly overwhelmed the empire, forcing the Jin court to retreat and entrench itself in the south past the Yangtze river, starting what is known as the Eastern Jin dynasty around 317. Towards the end of the century, Emperor of the Former Qin, Fu Jin, united the north under his banner, and planned to conquer the Jin dynasty in the south, so as to finally reunite the land, but was decisively defeated at the Battle of Fei River in 383, causing massive unrest and civil war in his empire, thereby leading to the fall of the Former Qin, and the continued existence of the Eastern Jin dynasty.According to archaeologists, sufficient archaeological evidence correlates of state-level societies coalesced in the 4th century to show the existence in Korea of the Three Kingdoms (300/400668 CE) of Baekje, Goguryeo, and Silla.Historians of the Roman Empire refer to the "Long Fourth Century" to the period spanning the fourth century proper but starting earlier with the accession of the Emperor Diocletian in 284 and ending later with the death of Honorius in 423 or of Theodosius II in 450.[3]See also: Christianity in the 4th centuryGregory the Illuminator mosaic, converted Armenia from Zoroastrianism to ChristianityContemporary bronze head of Constantine I (r. 306337 AD)Early 4th century Former audience hall now known as the Basilica, Trier, Germany, is built.Early 4th century The Gupta Empire is established.301: Armenia first to adopt Christianity as state religion.304439: The Sixteen Kingdoms in China begins.306337: Constantine the Great, ends persecution of Christians in the Roman Empire (see also Constantinian shift) and Constantinople becomes new seat of government (New Rome).Tikal had a population of about 100,000 when it was conquered by Teotihuacan, less than a fourth of its peak population[4]320: Butuan Boat One, the oldest known Balangay, a multi-purpose ship native to the Philippines is built.325328: The Kingdom of Aksum adopts Christianity.325: Constantine the Great calls the First Council of Nicaea to pacify Christianity in the grip of the Arian controversy.335380: Samudragupta expands the Gupta Empire.337: Constantine the Great is baptized a Christian on his death bed.350: About this time the Kingdom of Aksum conquers the Kingdom of Kush.350400: At some time during this period, the Huns began to attack the Sassanid Empire.[2]350: The Kutai Martadipura kingdom in eastern Borneo produced the earliest known stone inscriptions in Indonesia known as the Mulavarman inscription written in the Sanskrit language using Pallava scripture.[5]Mid-4th century Dish, from Mildenhall, England, is made. It is now kept at the British Museum, London.Mid-4th century Wang Xizhi makes a portion of a letter from the Feng Ju album. Six Dynasties period. It is now kept at National Palace Museum, Taipei, Taiwan, Republic of China.365: An earthquake with a magnitude of at least eight strikes the Eastern Mediterranean. The following tsunami causes widespread destruction in Crete, Greece, Libya, Egypt, Cyprus, and Sicily.376: Visigoths appear on the Danube and are allowed entry into the Roman Empire in their flight from the Huns.378: Battle of Adrianople: Roman army is defeated by the Visigoth cavalry. Emperor Valens is killed.378395: Theodosius I, Roman emperor, bans pagan worship, Christianity is made the official religion of the Empire.378: Siyaj K'ak' conquers Waka on (January 8), Tikal (January 16) and Uaxactun.Wall painting of the Council of Constantinople (381) in the Stavropoleos monastery, Romania381: First Council of Constantinople reaffirms the Christian doctrine of the Trinity by adding to the creed of Nicaea.383: Battle of Fei River in China.395: The Battle of Canhe Slope occurs.395: Roman emperor Theodosius I dies, causing the Roman Empire to split permanently.Late 4th century: Cubiculum of Leonis, Catacomb of Commodilla, near Rome, is made.Late 4th century: Atrium added in the Old St. Peter's Basilica, Rome.For a more comprehensive list, see Timeline of historic inventions 4th century.The Stirrup was invented in China, no later than 322.[6][1]Kama Sutra, dated between c.400 BC to c. 300 AD.[7][8]Iron pillar of Delhi, India is the world's first Iron Pillar.[citation needed]Trigonometric functions: The trigonometric functions sine and versine originated in Indian astronomy.[9]Codex Sinaiticus and the Codex Vaticanus Graecus 1209, are the earliest Christian bibles.[10][11]Book of Steps, Syriac religious discourses.[citation needed]^ a b "The invention and influences of stirrup". Archived from the original on December 3, 2008.^ a b Roberts, J: "History of the World". Penguin, 1994.^ The Long Fourth Century 284450: Continuity and Change in the Later Roman Empire ed. S. McGill, C. Sogno and E. Watts (Cambridge 2008).^ "The Maya: Glory and Ruin". National Geographic Magazine. Archived from the original on April 9, 2008.^ "The Austronesians: Historical and Comparative Perspectives". ANU Press. Archived from the original on 2013-12-25. Retrieved 2013-04-29.^ Lee, Adela C.Y. "The stirrup and its effect on chinese military history". Silkroad Foundation.^ Sengupta, J. (2006). Refractions of Desire, Feminist Perspectives in the Novels of Toni Morrison, Michle Roberts, and Anita Desai. Atlantic Publishers & Distributors. p.21. ISBN978-81-269-0629-1. Archived from the original on 4 May 2016. Retrieved 7 December 2014.^ Kakar, Sudhir; Doniger, Wendy (2003). Kamasutra. Oxford; Toronto: Oxford University Press. pp.xi. ISBN978-0-19-283982-4.^ Bag, A.K. (1979). Mathematics In Ancient and Medieval India. Delhi: Chaukhambha Orientalia. p.15.^ Aland, Kurt; Aland, Barbara (1995). The Text of the New Testament: An Introduction to the Critical Editions and to the Theory and Practice of Modern Textual Criticism. Erroll F. Rhodes (trans.). Grand Rapids, Michigan: William B. Eerdmans Publishing Company. p.109. ISBN978-0-8028-4098-1.^ "Liste Handschriften". Mnster: Institute for New Testament Textual Research. Retrieved 16 March 2013.Retrieved from " 4The following pages link to 4th century External tools(link counttransclusion countsorted list) See help page for transcluding these entriesShowing 50 items.View (previous 50 | next 50) (20 | 50 | 100 | 250 | 500)List of decades, centuries, and millennia (links | edit)Religion in pre-Islamic Arabia (links | edit)Rosetta Stone (links | edit)20th century (links | edit)15th century (links | edit)16th century (links | edit)17th century (links | edit)18th century (links | edit)14th century (links | edit)1st century (links | edit)13th century (links | edit)397 (links | edit)12th century (links | edit)11th century (links | edit)7th century (links | edit)10th century (links | edit)9th century (links | edit)8th century (links | edit)6th century (links | edit)5th century (links | edit)3rd century (links | edit)2nd century (links | edit)405 (links | edit)4th century BC (links | edit)432 (links | edit)200s (decade) (links | edit)300s (decade) (links | edit)1st century BC (links | edit)2nd century BC (links | edit)3rd century BC (links | edit)5th century BC (links | edit)6th century BC (links | edit)400s (decade) (links | edit)310s (links | edit)320s (links | edit)321 (links | edit)21st century BC (links | edit)11th century BC (links | edit)286 (links | edit)476 (links | edit)385 (links | edit)7th century BC (links | edit)410 (links | edit)325 (links | edit)380s (links | edit)381 (links | edit)470s (links | edit)430s (links | edit)430 (links | edit)510s (links | edit)View (previous 50 | next 50) (20 | 50 | 100 | 250 | 500)Retrieved from " WhatLinksHere/4th_century"Patient Information Leafletshair-loss-female-pattern-androgenetic-alopeciaWillan House, 4 Fitzroy Square, London, W1T 5HQ|+44 (0)020 7383 02662025 British Association of DermatologistsOverviewCausesOther conditionsTreatmentPreventionFAQSummaryFemale pattern baldness, or androgenetic alopecia, is a type of hair loss that occurs primarily due to genetics. It typically causes general hair thinning, or for the parting to get wider.In males, androgenetic alopecia causes a receding hairline. In females, the hair loss tends to be more diffuse. The condition is common in females, particularly after menopause.In this article, we look at the causes and risk factors for female pattern baldness, as well as treatment and prevention.Share on PinterestSouth_agency/Getty ImagesFemale pattern baldness is a type of hair loss. The medical name for it is androgenetic alopecia. The condition affects all sexes, but it appears differently in females than in males.Males who have androgenetic alopecia tend to develop a receding hairline and bald spots. Females with the condition usually experience general hair thinning instead. The scalp may be visible, but the hairline usually does not recede.Shedding about 50 to 100 hairs a day is typical, but new growth will usually replace these hairs. However, if someone has female pattern baldness, the hair does not grow back.The first sign of female pattern hair loss may be a widening part, or thinning on the crown of the head. A person may also notice less hair around the temples, but generally, the hairline will remain the same.As female pattern hair loss progresses, thinning on the crown of the head may become more apparent. Without treatment, a person may lose a significant amount of hair in this area. However, not everyone experiences this.Female pattern baldness is primarily a genetic condition. People can inherit the genes that cause it from either parent. These genes cause hair follicles to respond more strongly to androgens, or male sex hormones.Everyone has male sex hormones, including females. They play a role in regulating hair growth, which occurs in a cycle of four phases:anagen phase: hair growscatagen phase: hair growth slowstelogen phase: hair restsexogen phase: hair falls outAndrogen hormones stop hair growth, so an increased response to these hormones results in a shorter anagen phase. Over time, this leads to the hair becoming shorter and thinner.Other factors that may play a role in female pattern hair loss include:Female pattern baldness tends to be more common after menopause. This may be because, during perimenopause, estrogen levels decline. This suggests that estrogen may stimulate hair growth or protect against hair loss.Currently, this is only a theory. Scientists are still learning about the role estrogen plays in the hair growth cycle.Another point in life when a person is more likely to develop female pattern hair loss is after giving birth. Again, this may be because estrogen levels are lower during this time.An example of this is polycystic ovary syndrome (PCOS), a common condition that causes higher levels of androgens in females. Although hair growth is one of the symptoms of PCOS, it may also lead to hair loss.A 2020 study involving 472 women with female pattern hair loss showed that 38 already had a PCOS diagnosis. Researchers then discovered PCOS in an additional 12 women. In total, this accounted for 10.5% of the study participants.While androgenetic alopecia is a common cause of hair loss in females, there are other factors and medical conditions that can cause it. These include:Nutritional deficiencies: Several nutritional deficiencies can result in hair loss, including iron, zinc, protein, and biotin deficiencies. Iron deficiency, in particular, affects more women than men, particularly during pregnancy or as a result of heavy periods.Stress: Periods of intense or long-term stress can contribute to hair loss. When stress is the main cause, reducing it can allow the hair to grow back to its usual thickness in 6 to 9 months, according to the American Academy of Dermatology Association (AAD).Traction alopecia: Traction alopecia is hair loss that occurs when a person frequently wears hairstyles that pull the hair too tightly. Similarly, harsh chemical treatments can also lead to hair loss.Scalp infections or skin conditions: Infections of the scalp or hair follicles, as well as conditions such as psoriasis, can inflame the area and lead to hair loss.Thyroid disease: Problems with the thyroid, or thyroid disease, can cause hair thinning. Without treatment, it may become noticeable hair loss.Sexually transmitted infection (STI): Some STIs, such as syphilis, can lead to hair loss if left untreated.Alopecia areata: Alopecia areata is an autoimmune disorder that causes the immune system to attack the hair follicles, causing hair to fall out.Medical treatments: Certain medications, such as chemotherapy drugs, can cause hair loss as a side effect. However, hair usually regrows once a person has stopped taking the medication.Treatment for female pattern baldness can help prevent future hair loss and may result in some hair regrowth. The options include:Minoxidil is a topical treatment that people need to apply to the scalp every day. It may stimulate hair growth, as well as prevent further hair thinning. However, it can take 4 to 6 months of regular use to produce visible results.Side effects can include an initial period of hair shedding, skin irritation, or dryness. Hair loss may return after a person stops using the product.Finasteride is an oral medication people can take alone or while using minoxidil on the skin at the same time. However, the effectiveness of finasteride in females is uncertain, and it is not suitable for people who may become pregnant, as it affects fetal development.One of the common oral medications for female pattern hair loss is spironolactone, which is an antiandrogen drug. Spironolactone can cause side effects, such as:irregular periodsdizzinessheadacherashthe growth of fine, silky, light-colored hair on the facehigh potassium levelsHowever, these side effects can improve on their own.Laser or red light therapy is another treatment that may help with female pattern hair loss. People can buy these devices over the counter to use at home or get professional treatments.A 2020 review of previous research showed low-level light therapy (LLLT) treatment led to a significant increase in hair density and diameter. The research review involved a mixed population with three studies involving women only.Some people reported minor side effects, such as scalp irritation, itchiness, or a warm sensation, but these went away when the treatment was over.However, while there is evidence LLLT is effective, some previous studies have had relationships with companies in the industry, so the research may be biased.Hair transplants involve taking healthy hair from another part of the body and transplanting it to the scalp. It is an effective treatment, but it requires people to have enough donor hair for the procedure to work. It takes some time for a person to see results after a hair transplant. This often occurs in around 6 to 9 months, according to the AAD, but it may take up to a year.Learn more about hair transplants.As female pattern hair loss is largely genetic, it is not always preventable. However, taking certain steps can help people keep their hair as healthy as possible, including:washing the hair gently with a mild shampoo and conditionerusing detangling spray or leave-in conditioner before brushingusing the lowest setting on hair dryers to dry the hairreducing exposure to heat from straighteners, curling irons, and other deviceslimiting chemical hair treatments such as perms or relaxerseating a balanced diet containing enough calories, protein, vitamins, and mineralsstopping smoking if relevantBelow are some answers to frequently asked questions about female pattern baldness.Some treatments for female pattern baldness can stimulate new hair growth, which may reverse some of the effects. However, the results vary from person to person. Starting treatment as early as possible may prevent hair loss from progressing.Female pattern baldness is very common. Androgenetic alopecia may affect up to 50% of males and females.Female pattern baldness can start at any age, but it typically begins around midlife during a persons 40s, 50s, or 60s.There is no single treatment that works best for all people with female pattern hair loss. There are several effective options, but what is suitable for each individual will depend on their circumstances. For example, some people may respond well to medications, while others may have side effects and prefer laser therapy. People can talk with a dermatologist to help determine the best choice for them.Female pattern baldness, or androgenetic alopecia, is a common form of hair loss. In females, the hair loss usually involves general thinning, particularly around the crown. The hair parting may grow wider, but the hairline typically remains the same.Female pattern hair loss is mainly genetic, but there are things people can do to help slow or prevent the hair loss. Several treatments, including medications and laser therapy, can provide results, but they take time to work.For some, hair loss is distressing and may affect their self-confidence. People who have concerns about hair loss can talk with a doctor or another healthcare professional, who can help determine the cause and best treatment options.DermatologySeniors / AgingWomen's Health / GynecologyThis article needs more reliable medical references for verification or relies too heavily on primary sources. Please review the contents of the article and add the appropriate references if you can. Unsourced or poorly sourced material may be challenged and removed. Find sources:"Pattern hair loss"news newspapers books scholar JSTOR (February 2025)Medical conditionPattern hair lossOther namesMale pattern baldness;female pattern baldness;androgenic alopecia;androgenetic alopecia; alopecia androgeneticaMale-pattern hair loss shown on the vertex of the scalpSpecialtyDermatology, plastic surgeryPattern hair loss (also known as androgenetic alopecia (AGA)[1]) is a hair loss condition that primarily affects the top and front of the scalp.[2][3] In male-pattern hair loss (MPHL), the hair loss typically presents itself as either a receding front hairline, loss of hair on the crown and vertex of the scalp, or a combination of both. Female-pattern hair loss (FPHL) typically presents as a diffuse thinning of the hair across the entire scalp.[3] The condition is caused by a combination of male sex hormones (balding never occurs in castrated men) and genetic factors.[4]Some research has found evidence for the role of oxidative stress in hair loss,[5] the microbiome of the scalp,[6][7] genetics, and circulating androgens; particularly dihydrotestosterone (DHT).[3] Men with early onset androgenic alopecia (before the age of 35) have been deemed the male phenotypic equivalent for polycystic ovary syndrome (PCOS).[8][9][10][11] The cause in female pattern hair loss remains unclear;[3] androgenetic alopecia for women is associated with an increased risk of polycystic ovary syndrome (PCOS).[12][13][14]Management may include simply accepting the condition[3] or shaving one's head to improve the aesthetic aspect of the condition.[15] Otherwise, common medical treatments include minoxidil, finasteride, dutasteride, or hair transplant surgery.[3] Use of finasteride and dutasteride in women is not well-studied and may result in birth defects if taken during pregnancy.[3]By the age of 50, pattern hair loss affects about half of males and a quarter of females.[3] It is the most common cause of hair loss. Both males aged 4091[16] and younger male patients of early onset AGA (before the age of 35) had a higher likelihood of metabolic syndrome (MetS)[17][18][19][20] and insulin resistance.[21] With younger males, studies found metabolic syndrome to be at approximately a 4 increased frequency, which is deemed clinically significant.[22][23] Abdominal obesity, hypertension, and lowered high density lipoprotein were also significantly higher for younger groups.[24]Hair follicle and mesenchymal dermal papilla, labelled at topPattern hair loss is classified as a form of non-scarring hair loss.[citation needed]Male-pattern hair loss begins above the temples and at the vertex (calvaria) of the scalp. As it progresses, a rim of hair at the sides and rear of the head remains. This has been referred to as a "Hippocratic wreath" and rarely progresses to complete baldness.[25]Female-pattern hair loss more often causes diffuse thinning without hairline recession; similar to its male counterpart, female androgenic alopecia rarely leads to total hair loss.[26] The Ludwig scale grades severity of female-pattern hair loss. These include Grades 1, 2, 3 of balding in women based on their scalp showing in the front due to thinning of hair.[27]In most cases, receding hairline is the first starting point; the hairline starts moving backwards from the front of the head and the sides.[28]The cause of pattern hair loss is not yet fully understood. It appears to be the result of genetic changes that make the activity of hair follicles on the scalp become sensitive to the presence of androgenic hormones, cholesterol, and proteins such as insulin-like growth factor.[citation needed]KRT37 is the only keratin that is regulated by androgens.[29] This sensitivity to androgens was acquired by Homo sapiens and is not shared with their great ape cousins. Although Winter et al. found that KRT37 is expressed in all the hair follicles of chimpanzees, it was not detected in the head hair of modern humans. As androgens are known to grow hair on the body but decrease it on the scalp, this lack of scalp KRT37 may help explain the paradoxical nature of Androgenic alopecia as well as the fact that head hair anagen cycles are extremely long.[29]Although it is generally accepted that male pattern baldness follows a pattern of autosomal dominant inheritance, more recent research has shown that approximately 80% of bald men have bald fathers. This is greater than would be expected if pattern balding were a purely autosomal trait, and may suggest that there is an important paternal route of inheritance, either through a Y-chromosome gene or a paternal imprinting effect.[30]Androgens can interact with the Wnt signalling pathway to cause hair lossThe initial programming of pilosebaceous units of hair follicles begins in utero.[31] The physiology is primarily androgenic, with dihydrotestosterone (DHT) being the major contributor at the dermal papillae. Men with premature androgenic alopecia tend to have lower than normal values of sex hormone-binding globulin (SHBG), follicle stimulating hormone (FSH), testosterone, and epitestosterone when compared to men without pattern hair loss.[11] Although hair follicles were previously thought to be permanently gone in areas of complete hair loss, they are more likely dormant, as recent studies have shown the scalp contains the stem cell progenitor cells from which the follicles arose.[32][33][non-primary source needed]Transgenic studies have shown that growth and dormancy of hair follicles are related to the activity of insulin-like growth factor (IGF) at the dermal papillae, which is affected by DHT. Androgens are important in male sexual development around birth and at puberty. They regulate sebaceous glands, apocrine hair growth, and libido. With increasing age, androgens stimulate hair growth on the face, but can suppress it at the temples and scalp vertex, a condition that has been referred to as the 'androgen paradox'.[34]Men with androgenic alopecia typically have higher 5-reductase, higher total testosterone, higher unbound/free testosterone, and higher free androgens, including DHT.[35] 5-alpha-reductase converts free testosterone into DHT, and is highest in the scalp and prostate gland. DHT is most commonly formed at the tissue level by 5-reduction of testosterone.[36] The genetic corollary that codes for this enzyme has been discovered.[37] Prolactin has also been suggested to have different effects on the hair follicle across gender.[38]Also, crosstalk occurs between androgens and the Wnt-beta-catenin signaling pathway that leads to hair loss. At the level of the somatic stem cell, androgens promote differentiation of facial hair dermal papillae, but inhibit it at the scalp.[34] Other research suggests the enzyme prostaglandin D2 synthase and its product prostaglandin D2 (PGD2) in hair follicles as contributive.[39]These observations have led to study at the level of the mesenchymal dermal papillae.[40] Types 1 and 2 5 reductase enzymes are present at pilosebaceous units in papillae of individual hair follicles.[41] They catalyze formation of the androgen dihydrotestosterone from testosterone, which in turn regulate hair growth.[34] Androgens have different effects at different follicles: they stimulate IGF-1 at facial hair, leading to growth, but can also stimulate TGF 1, TGF 2, dickkopf1, and IL-6 at the scalp, leading to catagenic miniaturization.[34] Hair follicles in anaphase express four different caspases. Significant levels of inflammatory infiltrate have been found in transitional hair follicles.[42] Interleukin 1 is suspected to be a cytokine mediator that promotes hair loss.[43]The fact that hair loss is cumulative with age while androgen levels fall as well as the fact that finasteride does not reverse advanced stages of androgenetic alopecia remains a mystery, but possible explanations are higher conversion of testosterone to DHT locally with age as higher levels of 5-alpha reductase are noted in balding scalp, and higher levels of DNA damage in the dermal papilla as well as senescence of the dermal papilla due to androgen receptor activation and environmental stress.[44]Multiple cross-sectional studies have found associations between early androgenic alopecia, insulin resistance, and metabolic syndrome,[45][46] with low HDL being the component of metabolic syndrome with highest association.[47] Linolenic and linoleic acids, two major dietary sources of HDL, are 5 alpha reductase inhibitors.[48] Premature androgenic alopecia and insulin resistance may be a clinical constellation that represents the male homologue, or phenotype, of polycystic ovary syndrome.[49] Others have found a higher rate of hyperinsulinemia in family members of women with polycystic ovarian syndrome.[50] With early-onset AGA having an increased risk of metabolic syndrome, poorer metabolic profiles are noticed in those with AGA, including metrics for body mass index, waist circumference, fasting glucose, blood lipids, and blood pressure.[51]In support of the association, finasteride improves glucose metabolism and decreases glycated hemoglobin HbA1c, a surrogate marker for diabetes mellitus.[52] The low SHBG seen with premature androgenic alopecia is also associated with, and likely contributory to, insulin resistance,[53] and for which it still is used as an assay for pediatric diabetes mellitus.[54]Obesity leads to upregulation of insulin production and decrease in SHBG. Further reinforcing the relationship, SHBG is downregulated by insulin in vitro, although SHBG levels do not appear to affect insulin production.[55] In vivo, insulin stimulates both testosterone production and SHBG inhibition in normal and obese men.[56] The relationship between SHBG and insulin resistance has been known for some time; decades prior, ratios of SHBG and adiponectin were used before glucose to predict insulin resistance.[57] Patients with Laron syndrome, with resultant deficient IGF, demonstrate varying degrees of alopecia and structural defects in hair follicles when examined microscopically.[58]Because of its association with metabolic syndrome and altered glucose metabolism, both men and women with early androgenic hair loss should be screened for impaired glucose tolerance and diabetes mellitus II.[11] Measurement of subcutaneous and visceral adipose stores by MRI, demonstrated inverse association between visceral adipose tissue and testosterone/DHT, while subcutaneous adipose correlated negatively with SHBG and positively with estrogen.[59] SHBG association with fasting blood glucose is most dependent on intrahepatic fat, which can be measured by MRI in and out of phase imaging sequences. Serum indices of hepatic function and surrogate markers for diabetes, previously used, show less correlation with SHBG by comparison.[60]Female patients with mineralocorticoid resistance present with androgenic alopecia.[61]IGF levels have been found lower in those with metabolic syndrome.[62] Circulating serum levels of IGF-1 are increased with vertex balding, although this study did not look at mRNA expression at the follicle itself.[63] Locally, IGF is mitogenic at the dermal papillae and promotes elongation of hair follicles. The major site of production of IGF is the liver, although local mRNA expression at hair follicles correlates with increase in hair growth. IGF release is stimulated by growth hormone (GH). Methods of increasing IGF include exercise, hypoglycemia, low fatty acids, deep sleep (stage IV REM), estrogens, and consumption of amino acids such as arginine and leucine. Obesity and hyperglycemia inhibit its release. IGF also circulates in the blood bound to a large protein whose production is also dependent on GH. GH release is dependent on normal thyroid hormone. During the sixth decade of life, GH decreases in production. Because growth hormone is pulsatile and peaks during sleep, serum IGF is used as an index of overall growth hormone secretion. The surge of androgens at puberty drives an accompanying surge in growth hormone.[64]The expression of insulin resistance and metabolic syndrome, AGA is related to being an increased risk factor for cardiovascular diseases, glucose metabolism disorders,[65] type 2 diabetes,[66][67] and enlargement of the prostate.[68]A number of hormonal changes occur with aging:Decrease in testosteroneDecrease in serum DHT and 5-alpha reductaseDecrease 3AAG, a peripheral marker of DHT metabolismIncrease in SHBGDecrease in androgen receptors, 5-alpha reductase type I and II activity, and aromatase in the scalp[69][70]This decrease in androgens and androgen receptors, and the increase in SHBG are opposite the increase in androgenic alopecia with aging. This is not intuitive, as testosterone and its peripheral metabolite, DHT, accelerate hair loss, and SHBG is thought to be protective. The ratio of T/SHBG, DHT/SHBG decreases by as much as 80% by age 80, in numeric parallel to hair loss, and approximates the pharmacology of antiandrogens such as finasteride.[71]Free testosterone decreases in men by age 80 to levels double that of a woman at age 20. About 30% of normal male testosterone level, the approximate level in females, is not enough to induce alopecia; 60%, closer to the amount found in elderly men, is sufficient.[72] The testicular secretion of testosterone perhaps "sets the stage" for androgenic alopecia as a multifactorial diathesis stress model, related to hormonal predisposition, environment, and age. Supplementing eunuchs with testosterone during their second decade, for example, causes slow progression of androgenic alopecia over many years, while testosterone late in life causes rapid hair loss within a month.[73]An example of premature age effect is Werner's syndrome, a condition of accelerated aging from low-fidelity copying of mRNA. Affected children display premature androgenic alopecia.[74]The diagnosis of androgenic alopecia can be usually established based on clinical presentation in men. In women, the diagnosis usually requires more complex diagnostic evaluation. Further evaluation of the differential requires exclusion of other causes of hair loss, and assessing for the typical progressive hair loss pattern of androgenic alopecia.[75] Trichoscopy can be used for further evaluation.[76] Biopsy may be needed to exclude other causes of hair loss,[77] and

histology would demonstrate perifollicular fibrosis.[78][79] The HamiltonNorwood scale has been developed to grade androgenic alopecia in males by severity.[citation needed]Main article: Management of hair lossCombinations of finasteride, minoxidil and ketoconazole are more effective than individual use.[80]Combination therapy of LLLT or microneedling with finasteride[81] or minoxidil demonstrated substantive increases in hair count.[82]Finasteride is a medication of the 5-reductase inhibitors (5-ARIs) class.[83] By inhibiting type II 5-AR, finasteride prevents the conversion of testosterone to dihydrotestosterone in various tissues including the scalp.[83][84] Increased hair on the scalp can be seen within three months of starting finasteride treatment and longer-term studies have demonstrated increased hair on the scalp at 24 and 48 months with continued use.[84] Treatment with finasteride more effectively treats male-pattern hair loss at the crown than male-pattern hair loss at the front of the head and temples.[84]Dutasteride is a medication in the same class as finasteride but inhibits both type I and type II 5-alpha reductase.[84] Dutasteride is approved for the treatment of male-pattern hair loss in Korea and Japan, but not in the United States.[84] However, it is commonly used off-label to treat male-pattern hair loss.[84]Minoxidil dilates small blood vessels; it is not clear how this causes hair to grow.[85] Other treatments include tretinoin combined with minoxidil, ketoconazole shampoo, dermarolling (Collagen induction therapy), spironolactone,[86] alfatradiol, topilutamide (fluridil),[83] topical melatonin,[87][88][89] and intradermal and intramuscular botulinum toxin injections to the scalp.[90]Progressive stages of female pattern hair lossThere is evidence supporting the use of minoxidil as a safe and effective treatment for female pattern hair loss, and there is no significant difference in efficiency between 2% and 5% formulations.[91] Finasteride was shown to be no more effective than placebo based on low-quality studies.[91] The effectiveness of laser-based therapies is unclear.[91] Bicalutamide, an antiandrogen, is another option for the treatment of female pattern hair loss.[92][5][93]More advanced cases may be resistant or unresponsive to medical therapy and require hair transplantation. Naturally occurring units of one to four hairs, called follicular units, are excised and moved to areas of hair restoration.[86] These follicular units are surgically implanted in the scalp in close proximity and in large numbers. The grafts are obtained from either follicular unit transplantation (FUT) or follicular unit extraction (FUE). In the former, a strip of skin with follicular units is extracted and dissected into individual follicular unit grafts, and in the latter individual hairs are extracted manually or robotically. The surgeon then implants the grafts into small incisions, called recipient sites.[94][95] Cosmetic scalp tattoos can also mimic the appearance of a short, buzzed haircut.Low-level laser therapy or photobiomodulation is also referred to as red light therapy and cold laser therapy. It is a non-invasive treatment option.[citation needed]LLLT is shown to increase hair density and growth in both genders. The types of devices (hat, comb, helmet) and duration did not alter the effectiveness,[96] with more emphasis to be placed on lasers compared to LEDs.[97] Ultraviolet and infrared light are more effective for alopecia areata, while red light and infrared light is more effective for androgenic alopecia.[98]Medical reviews suggest that LLLT is as effective or potentially more than other non invasive and traditional therapies like minoxidil and finasteride but further studies such as RCTs, long term follow up studies, and larger double blinded trials need to be conducted to confirm the initial findings.[99][81][100]Using ones own cells and tissues and without harsh side effects, PRP is beneficial for alopecia areata[101] and androgenic alopecia and can be used as an alternative to minoxidil or finasteride.[102] It has been documented to improve hair density and thickness in both genders.[103] A minimum of 3 treatments, once a month for 3 months are recommended, and afterwards a 3-6 month period of continual appointments for maintenance.[104] Factors that determine efficacy include amount of sessions, double versus single centrifugation, age and gender, and where the PRP is inserted.[105]Future larger randomized controlled trials and other high quality studies are still recommended to be carried out and published for a stronger consensus.[99][103][106] Further development of a standardized practice for procedure is also recommended.[101]Many people use unproven treatments.[107] Regarding female pattern alopecia, there is no evidence for vitamins, minerals, or other dietary supplements.[108] As of 2008, there is little evidence to support the use of lasers to treat male-pattern hair loss.[109] The same applies to special lights.[108] Dietary supplements are not typically recommended.[109] A 2015 review found a growing number of papers in which plant extracts were studied but only one randomized controlled clinical trial, namely a study in 10 people of saw palmetto extract.[110][111]A 2023 study on genetically engineered mice published in the journal PNAS found that increasing production of a particular microRNA in hair follicle stem cells, which naturally harden with age, softened the cells and stimulated hair growth. The authors of the study said the next research step is to introduce the microRNA into the stem cells using nanoparticles applied directly to the skin, with the goal of developing a similar topical application for humans.[112]Androgenic alopecia is typically experienced as a "moderately stressful condition that diminishes body image satisfaction".[113] However, although most men regard baldness as an unwanted and distressing experience, they usually are able to cope and retain integrity of personality.[114]Although baldness is not as common in women as in men, the psychological effects of hair loss tend to be much greater. Typically, the frontal hairline is preserved, but the density of hair is decreased on all areas of the scalp. Previously, it was believed to be caused by testosterone just as in male baldness, but most women who lose hair have normal testosterone levels.[115]Female androgenic alopecia has become a growing problem that, according to the American Academy of Dermatology, affects around 30million women in the United States. Although hair loss in females normally occurs after the age of 50 or even later when it does not follow events like pregnancy, chronic illness, crash diets, and stress among others, it is now occurring at earlier ages with reported cases in women as young as 15 or 16.[116]For male androgenic alopecia, by the age of 50 30-50% of men have it, hereditarily there is an 80% predisposition.[117] Notably, the link between androgenetic alopecia and metabolic syndrome is strongest in non-obese men.[118]Certain studies have suggested androgenic alopecia conveys survival advantageStudies have been inconsistent across cultures regarding how balding men rate on the attraction scale. While a 2001 South Korean study showed that most people rated balding men as less attractive,[119] a 2002 survey of Welsh women found that they rated bald and gray-haired men quite desirable.[120] One of the proposed social theories for male pattern hair loss is that men who embraced complete baldness by shaving their heads subsequently signaled dominance, high social status, and/or longevity.[15]Biologists have hypothesized the larger sunlight-exposed area would allow more vitamin D to be synthesized, which might have been a "finely tuned mechanism to prevent prostate cancer" as the malignancy itself is also associated with higher levels of DHT.[121]An ancient phenomenon: Greek philosophers with and without much hair (from left to right: Socrates, Antisthenes, Chrysippus, and Epicurus, fifth to third centuries BC)Many myths exist regarding the possible causes of baldness and its relationship with one's virility, intelligence, ethnicity, job, social class, wealth, and many other characteristics.[citation needed]Because it increases testosterone levels, many Internet forums[which?] have put forward the idea that weight training and other forms of exercise increase hair loss in predisposed individuals. Although scientific studies do support a correlation between exercise and testosterone, no direct study has found a link between exercise and baldness. However, a few have found a relationship between a sedentary life and baldness, suggesting exercise is causally relevant. The type or quantity of exercise may influence hair loss.[122][123]Testosterone levels are not a good marker of baldness, and many studies actually show paradoxical low testosterone in balding persons, although research on the implications is limited.[citation needed]Emotional stress has been shown to accelerate baldness in genetically susceptible individuals.[124]Stress due to sleep deprivation in military recruits lowered testosterone levels, but is not noted to have affected SHBG.[125] Thus, stress due to sleep deprivation in fit males is unlikely to elevate DHT, which is one cause of male pattern baldness. Whether sleep deprivation can cause hair loss by some other mechanism is not clear.Levels of free testosterone are strongly linked to libido and DHT levels, but unless free testosterone is virtually nonexistent, levels have not been shown to affect virility. Men with androgenic alopecia are more likely to have a higher baseline of free androgens. However, sexual activity is multifactoral, and androgenic profile is not the only determining factor in baldness. Additionally, because hair loss is progressive and free testosterone declines with age, a male's hairline may be more indicative of his past than his present disposition.[126][127]Animal models of androgenic alopecia occur naturally and have been developed in transgenic mice;[128] chimpanzees (Pan troglodytes); bald uakaris (Cacajao rubicundus); and stump-tailed macaques (Macaca speciosa and M. arctoides). Of these, macaques have demonstrated the greatest incidence and most prominent degrees of hair loss.[129][130]Baldness is not a trait unique to human beings. One possible case study is about a maneless male lion in the Tsavo area. The Tsavo lion prides are unique in that they frequently have only a single male lion with usually seven or eight adult females, as opposed to four females in other lion prides. Male lions may have heightened levels of testosterone, which could explain their reputation for aggression and dominance, indicating that lack of mane may at one time have had an alpha correlation.[131]Although nonhuman primates do not go bald, their hairlines do undergo recession. In infancy the hairline starts at the top of the supraorbital ridge, but slowly recedes after puberty to create the appearance of a small forehead.[citation needed]^ "Androgenetic alopecia". National Library of Medicine, Bethesda, Maryland, United States. 1 August 2015. Archived from the original on 9 November 2020. Retrieved 3 April 2022.^ Wang EH, Monga I, Sallee BN, Chen JC, Abdelaziz AR, Perez-Lorenzo R, etal. (Jul 2022). "Primary cicatricial alopecias are characterized by dysregulation of shared gene expression pathways". PNAS Nexus. 1 (3): pgac111. doi:10.1093/pnasnexus/pgac111. PMC9308563. PMID35899069.^ a b c d e f g h Vary JC (November 2015). "Selected Disorders of Skin Appendages--Acne, Alopecia, Hyperhidrosis". The Medical Clinics of North America (Review). 99 (6): 11951211. doi:10.1016/j.mcna.2015.07.003. PMID26476248.^ Randall VA (2012-07-26). "Androgens and hair: a biological paradox with clinical consequences". Testosterone. Cambridge University Press. pp.154176. doi:10.1017/cbo9781139003353.008. ISBN978-1-139-00335-3. Male pattern baldness is androgen dependent, since it does not occur in castrates, unless they are given testosterone (Hamilton 1942), nor in XY individuals with androgen insensitivity due to non-functional androgen receptors (see Chapter 3). The genetic involvement in androgenetic alopecia is also pronounced.^ a b Meyer-Gonzalez T, Bacqueville D, Grimalt R, Mengeaud V, Piraccini BM, Rudnicka L, etal. (November 2021). "Current controversies in trichology: a European expert consensus statement". Journal of the European Academy of Dermatology and Venereology. 35 (Suppl 2): 311. doi:10.1111/jdv.17601. hdl:11585/863826. PMID34668238. S2CID239029062.^ Suzuki K, Inoue M, Cho O, Mizutani R, Shimizu Y, Nagahama T, etal. (2021). "Scalp Microbiome and Sebum Composition in Japanese Male Individuals with and without Androgenetic Alopecia". Microorganisms. 9 (10): 2132. doi:10.3390/microorganisms9102132. PMC8536999. PMID34683453.^ Huang J, Ran Y, Pradhan S, Yan W, Dai Y (2019). "Investigation on Microecology of Hair Root Fungi in Androgenetic Alopecia Patients". Mycopathologia. 184 (4): 505515. doi:10.1007/s11046-019-00345-8. PMID31240449. S2CID195353938.^ Cannarella R, La Vignera S, Condorelli RA, Calogero AE (2017). "Glycolipid and Hormonal Profiles in Young Men with Early-Onset Androgenetic Alopecia: A meta-analysis". Scientific Reports. 7 (1): 7801. Bibcode:2017NatSR...7.7801C. doi:10.1038/s41598-017-08528-3. PMC5552767. PMID28798373.^ Sanke S, Chander R, Jain A, Garg T, Yadav P (2016). "A Comparison of the Hormonal Profile of Early Androgenetic Alopecia in Men with the Phenotypic Equivalent of Polycystic Ovarian Syndrome in Women". JAMA Dermatology. 152 (9): 986991. doi:10.1001/jamadermatol.2016.1776. PMID27304785. S2CID26897074.^ Krysiak R, Kowalcze K, Okopie B (2022). "Impaired metabolic effects of metformin in men with early-onset androgenic alopecia". Pharmacological Reports. 74 (1): 216228. doi:10.1007/s43440-021-00347-8. PMC8786753. PMID34897595.^ a b c Starka L, Duskova M, Cermakova I, Vrbikov J, Hill M (December 2005). "Premature androgenic alopecia and insulin resistance. Male equivalent of polycystic ovary syndrome?". Endocrine Regulations. 39 (4). Slovak Academic Press: 127131. PMID16552990. Archived from the original (pdf) on March 8, 2019. Retrieved Mar 7, 2019.^ Goodman NF, Cobin RH, Futterweit W, Glueck JS, Legro RS, Carmina E, etal. (2015). "American Association of Clinical Endocrinologists, American College of Endocrinology, and Androgen Excess and Pcos Society Disease State Clinical Review: Guide to the Best Practices in the Evaluation and Treatment of Polycystic Ovary Syndrome--Part 1". Endocrine Practice. 21 (11): 12911500. doi:10.4158/EP15748.DSC. PMID26509855.^ Camacho-Martnez FM (2009). "Hair loss in women". Seminars in Cutaneous Medicine and Surgery. 28 (1): 1932. doi:10.1016/j.sder.2009.01.001 (inactive 2 December 2024). PMID19341939.{{cite journal}}: CS1 maint: DOI inactive as of December 2024 (link)^ Moura HH, Costa DL, Bagatin E, Sodr CT, Manela-Azulay M (2011). "Polycystic ovary syndrome: A dermatologic approach". Anais Brasileiros de Dermatologia. 86 (1): 111119. doi:10.1590/s0365-05962011000100015. PMID21437531.^ a b Dunn R (2012). "Why haven't bald men gone extinct?". New Scientist. 214 (2869): 4447. Bibcode:2012NewSc.214...44D. doi:10.1016/s0262-4079(12)61567-x. Archived from the original on June 27, 2012. Retrieved Dec 16, 2012.^ Su LH, Chen TH (2010). "Association of androgenetic alopecia with metabolic syndrome in men: A community-based survey". The British Journal of Dermatology. 163 (2): 371377. doi:10.1111/j.1365-2133.2010.09816.x. PMID20426781. S2CID23615726.^ Ertas R, Orscelik O, Kartal D, Dogan A, Ertas SK, Aydogdu EG, etal. (2016). "Androgenetic alopecia as an indicator of metabolic syndrome and cardiovascular risk". Blood Pressure. 25 (3): 141148. doi:10.3109/08037051.2015.1111021. PMID26585114. S2CID12031777.^ Dharam Kumar KC, Kishan Kumar YH, Neladimmanahally V (2018). "Association of Androgenetic Alopecia with Metabolic Syndrome: A Case-control Study on 100 Patients in a Tertiary Care Hospital in South India". Indian Journal of Endocrinology and Metabolism. 22 (2): 196199. doi:10.4103/ijem.IJEM_650_17. PMC5972473. PMID29911030.^ Bakry OA, Shoeib MA, El Shafiee MK, Hassan A (2014). "Androgenetic alopecia, metabolic syndrome, and insulin resistance: Is there any association? A case-control study". Indian Dermatology Online Journal. 5 (3): 276281. doi:10.4103/2229-5178.137776. PMC4144211. PMID25165643.^ Banger HS, Malhotra SK, Singh S, Mahajan M (2015). "Is Early Onset Androgenic Alopecia a Marker of Metabolic Syndrome and Carotid Artery Atherosclerosis in Young Indian Male Patients?". International Journal of Trichology. 7 (4): 141147. doi:10.4103/0974-7753.171566. PMC4738480. PMID26903742.^ Acibucu F, Kayatas M, Candan F (2010). "The association of insulin resistance and metabolic syndrome in early androgenetic alopecia". Singapore Medical Journal. 51 (12): 931936. PMID21221497.^ Sheikh FZ, Butt G, Hafeez R, Maqsood A, Altaf F, Hussain I (2021). "Association of Early-onset Androgenetic Alopecia and Metabolic Syndrome". Journal of the College of Physicians and Surgeons (Pakistan). 31 (2): 123127. doi:10.29271/jcpsp.2021.02.123. PMID33645175. S2CID232079822.^ Pengsalae N, Tanglertsampan C, Phichawong T, Lee S (2013). "Association of early-onset androgenetic alopecia and metabolic syndrome in Thai men: A case-control study". Journal of the Medical Association of Thailand = Chotmaihet Thangphaet. 96 (8): 947951. PMID23991602.^ Gopinath H, Upadya GM (2016). "Metabolic syndrome in androgenic alopecia". Indian Journal of Dermatology, Venereology and Leprology. 82 (4): 404408. doi:10.4103/0378-6323.174421. PMID27279298. S2CID24300999.^ "Hippocratic wreath (Baldness)". Britannica Online. Dec 15, 2012. Archived from the original on June 19, 2013. Retrieved Dec 15, 2012.^ "Female pattern baldness". MedlinePlus. Dec 15, 2012. Archived from the original on December 1, 2012. Retrieved Dec 15, 2012.^ Dinh QQ, Sinclair R (2006-08-08). "Female pattern hair loss: Current treatment concepts". Clinical Interventions in Aging. 2 (2). Dove Press: 189199. PMC2684510. PMID18044135.^ Rammos CK, Mardini S (January 2016). "Endoscopic Browlift in the Receding Hairline Patient". The Journal of Craniofacial Surgery. 27 (1): 156158. doi:10.1097/SCS.0000000000002266. PMID26674899. S2CID6206827.^ a b Jave-Suarez LF, Langbein L, Winter H, Praetzel S, Rogers MA, Schweizer J (March 2004). "Androgen regulation of the human hair follicle: the type I hair keratin hHa7 is a direct target gene in trichocytes". The Journal of Investigative Dermatology. 122 (3): 555564. doi:10.1111/j.0022-202X.2004.22336.x. PMID15086535.^ Treb RM, Tobin D (2 April 2010). Aging Hair. Springer Science & Business Media. p.16. ISBN978-3-642-02636-2. Archived from the original on 10 October 2024. Retrieved 17 May 2024.^ Alonso LC, Rosenfield RL (2003). "Molecular genetic and endocrine mechanisms of hair growth". Hormone Research. 60 (1): 113. doi:10.1159/000070821. PMID12792148. S2CID40910080.^ Garza LA, Yang CC, Zhao T, Blatt HB, Lee M, He H, etal. (February 2011). "Bald scalp in men with androgenetic alopecia retains hair follicle stem cells but lacks CD200-rich and CD34-positive hair follicle progenitor cells". The Journal of Clinical Investigation. 121 (2): 613622. doi:10.1172/JCI44478. PMC3026732. PMID21206086.^ Rahmani W, Sinha S, Biernaskie J (2020-05-11). "Immune modulation of hair follicle regeneration". npj Regenerative Medicine. 5 (1): 9. doi:10.1038/s41536-020-0095-2. PMC7214459. PMID32411394.^ a b c d Inui S, Itami S (March 2013). "Androgen actions on the human hair follicle: perspectives". Experimental Dermatology. 22 (3): 168171. doi:10.1111/exd.12024. PMID23016593. S2CID33521841.^ Zhang Y, Xu J, Jing J, Wu X, Lv Z (October 2018). "Serum Levels of Androgen-Associated Hormones Are Correlated with Curative Effect in Androgenic Alopecia in Young Men". Medical Science Monitor. 24: 77707777. doi:10.12659/MSM.913116. PMC6223099. PMID30376555.^ Kaufman JM, Vermeulen A (October 2005). "The decline of androgen levels in elderly men and its clinical and therapeutic implications". Endocrine Reviews. 26 (6): 833876. doi:10.1210/er.2004-0013. PMID15901667.^ Ellis JA, Panagiotopoulos S, Akdeniz A, Jerums G, Harrap SB (2005). "Androgenic correlates of genetic variation in the gene encoding 5alpha-reductase type 1". Journal of Human Genetics. 50 (10): 534537. doi:10.1007/s10038-005-0289-x. PMID16155734.^ Langan EA, Ramot Y, Goffin V, Griffiths CE, Foitzik K, Paus R (March 2010). "Mind the (gender) gap: does prolactin exert gender and/or site-specific effects on the human hair follicle?". The Journal of Investigative Dermatology. 130 (3): 886891. doi:10.1038/jid.2009.340. PMID19890346.^ Garza LA, Liu Y, Yang Z, Alagesan B, Lawson JA, Norberg SM, etal. (March 2012). "Prostaglandin D2 inhibits hair growth and is elevated in bald scalp of men with androgenetic alopecia". Science Translational Medicine. 4 (126): 126ra34. doi:10.1126/scitranslmed.3003122. PMC3319975. PMID22440736.^ Randall VA, Hibberts NA, Thornton MJ, Merrick AE, Hamada K, Kato S, etal. (2001). "Do androgens influence hair growth by altering the paracrine factors secreted by dermal papilla cells?". European Journal of Dermatology. 11 (4): 315320. PMID11399537.^ Bernard BA (1994). "[Molecular approach of hair biology]". Comptes Rendus des Sances de la Socit de Biologie et de ses Filiales. 188 (3): 223233. PMID7834505.^ Jaworsky C, Kligman AM, Murphy GF (September 1992). "Characterization of inflammatory infiltrates in male pattern alopecia: implications for pathogenesis". The British Journal of Dermatology. 127 (3): 239246. doi:10.1111/j.1365-2133.1992.tb00121.x. PMID1390168. S2CID11764980.^ Hoffmann R, Happle R (1995). "Does interleukin-1 induce hair loss?". Dermatology. 191 (4): 273275. doi:10.1159/000246567. PMID8573920.^ Yang YC, Fu HC, Wu CY, Wei KT, Huang KE, Kang HY (March 15, 2013). "Androgen receptor accelerates premature senescence of human dermal papilla cells in association with DNA damage". PLOS ONE. 8 (11): e79434. Bibcode:2013PLoSO...879434Y. doi:10.1371/journal.pone.0079434. PMC3828374. PMID24244503.^ Acibucu F, Kayatas M, Candan F (2010). "The association of insulin resistance and metabolic syndrome in early androgenetic alopecia". Singapore Medical Journal. 51 (12): 931936. PMID21221497.^ Gonzlez-Gonzlez JG, Mancillas-Adame LG, Fernndez-Reyes M, Gmez-Flores M, Lavalle-Gonzlez FJ, Ocampo-Candiani J, etal. (2009). "Androgenetic alopecia and insulin resistance in young men". Clinical Endocrinology. 71 (4): 494499. doi:10.1111/j.1365-2265.2008.03508.x. PMID19094069. S2CID205285087.^ Su LH, Chen TH (2010). "Association of androgenetic alopecia with metabolic syndrome in men: A community-based survey". British Journal of Dermatology. 163 (2): 371377. doi:10.1111/j.1365-2133.2010.09816.x. PMID20426781. S2CID23615726.^ Liang T, Liao S (1992). "Inhibition of steroid 5 alpha-reductase by specific aliphatic unsaturated fatty acids". The Biochemical Journal. 285 (Pt 2): 557562. doi:10.1042/bj2850557. PMC1132824. PMID1637346.^ Legro RS (2000). "Is there a male phenotype in polycystic ovary syndrome families?". Journal of Pediatric Endocrinology & Metabolism. 13 (Suppl 5): 13071309. PMID11117676.^ Norman RJ, Masters S, Hague W (1996). "Hyperinsulinemia is common in family members of women with polycystic ovary syndrome". Fertility and Sterility. 66 (6): 942947. doi:10.1016/s0015-0282(16)58687-7. PMID8941059.^ Qiu, Y., Zhou, X., Fu, S., Luo, S., & Li, Y. (2022). Systematic Review and Meta-analysis of the Association Between Metabolic Syndrome and Androgenetic Alopecia. Acta dermato-venereologica, 102, adv00645. Archived 2024-10-10 at the Wayback Machine^ Duskova M, Hill M, Starka L (2010). "Changes of metabolic profile in men treated for androgenetic alopecia with 1 mg finasteride". Endocrine Regulations. 44 (1): 38. doi:10.4149/endo_2010_01_3. PMID20151762.^ Pugeat M, Crave JC, Elmidani M, Nicolas MH, Garoscio-Cholet M, Lejeune H, etal. (1991). "Pathophysiology of sex hormone binding globulin (SHBG): Relation to insulin". The Journal of Steroid Biochemistry and Molecular Biology. 40 (46): 841849. doi:10.1016/0960-0760(91)90310-2. PMID1958579. S2CID248035.^ Gascn F, Valle M, Martos R, Ruz FJ, Ros R, Montilla P, etal. (2000). "Sex hormone-binding globulin as a marker for hyperinsulinemia and/or insulin resistance in obese children". European Journal of Endocrinology. 143 (1): 8589. doi:10.1530/eje.0.1430085. PMID10870035.^ Strain G, Zumoff B, Rosner W, Pi-Sunyer X (1994). "The relationship between serum levels of insulin and sex hormone-binding globulin in men: The effect of weight loss". The Journal of Clinical Endocrinology and Metabolism. 79 (4): 11731176. doi:10.1210/jcem.79.4.7962291. PMID7962291.^ Pasquali R, Casimirri F, De Iasio R, Mesini P, Boschi S, Chierici R, etal. (1995). "Insulin regulates testosterone and sex hormone-binding globulin concentrations in adult normal weight and obese men". The Journal of Clinical Endocrinology and Metabolism. 80 (2): 654658. doi:10.1210/jcem.80.2.7852532. PMID7852532.^ Ducluzeau PH, Cousin P, Malvoisin E, Bornet H, Vidal H, Laville M, etal. (2003). "Glucose-to-insulin ratio rather than sex hormone-binding globulin and adiponectin levels is the best predictor of insulin resistance in nonobese women with polycystic ovary syndrome". The Journal of Clinical Endocrinology and Metabolism. 88 (8): 36263631. doi:10.1210/jc.2003-030219. PMID12915646.^ Lurie R, Ben-Amitai D, Laron Z (2004). "Laron Syndrome (Primary Growth Hormone Insensitivity): A Unique Model to Explore the Effect of Insulin-Like Growth Factor 1 Deficiency on Human Hair". Dermatology. 208 (4): 314318. doi:10.1159/000077839. PMID15178913. S2CID8285817.^ Nielsen TL, Hagen C, Wraae K, Brixen K, Petersen PH, Haug E, etal. (2007). "Visceral and Subcutaneous Adipose Tissue Assessed by Magnetic Resonance Imaging in Relation to Circulating Androgens, Sex Hormone-Binding Globulin, and Luteinizing Hormone in Young Men". Journal of Clinical Endocrinology & Metabolism. 92 (7): 26962705. doi:10.1210/jc.2006-1847. PMID17426100.^ Bonnet F, Velayoudom Cephise FL, Gautier A, Dubois SV, Massart C, Camara A, etal. (2012). "Role of sex steroids, intra-hepatic fat and liver enzymes in the association between SHBG and metabolic features". Clinical Endocrinology. 79 (4): 517522. doi:10.1111/cen.12089. PMID23121021. S2CID24411342.^ Van Rossum EF, Lamberts SW (2006). "Glucocorticoid resistance syndrome: A diagnostic and therapeutic approach". Best Practice & Research Clinical Endocrinology & Metabolism. 20 (4): 611626. doi:10.1016/j.beem.2006.09.005. PMID17161335.^ Devarakonda K, Martha S, Pantam N, Thungathurthi S, Rao V (2008). "Study of insulin resistance in relation to serum IGF-I levels in subjects with different degrees of glucose tolerance". International Journal of Diabetes in Developing Countries. 28 (2): 5459. doi:10.4103/0973-3930.43100 (inactive 1 November 2024). PMC2772007. PMID19902049.{{cite journal}}: CS1 maint: DOI inactive as of November 2024 (link)^ Signorello LB, Wuu J, Hsieh C, Tzonou A, Trichopoulos D, Mantzoros CS (1999). "Hormones and hair patterning in men: A role for insulin-like growth factor 1?". Journal of the American Academy of Dermatology. 40 (2 Pt 1): 200203. doi:10.1016/s0190-9622(99)70188-x. PMID10025745.^ Rosenfeld RG (Nov 9, 2010). The IGF System: Molecular Biology, Physiology, and Clinical Applications (Contemporary Endocrinology). umana Press. ISBN978-1-61737-138-7.^ Duskova M, Starka L, Hill M, Dolezal M, Simunkova K, Cermakova I (2006). "Is there male androgenetic alopecia the sign of male equivalent of polycystic ovary syndrome or metabolic syndrome?". Endocrine Abstracts. 11 (11): 366. eISSN1479-6848. ISSN1470-3947. Archived from the original on 2023-10-14. Retrieved 2022-06-30.^ Cannarella R, Condorelli RA, Mongio LM, La Vignera S, Calogero AE (2018). "Does a male polycystic ovarian syndrome equivalent exist?". Journal of Endocrinological Investigation. 41 (1): 4957. doi:10.1007/s40618-017-0728-5. PMID28711970. S2CID11255317.^ Rodrguez-Gutirrez R, Salcido-Montenegro A, Gonzlez-Gonzlez JG (2018). "Early Clinical Expressions of Insulin Resistance: The Real Enemy to Look for". Diabetes Therapy: Research, Treatment and Education of Diabetes and Related Disorders. 9 (1): 435438. doi:10.1007/s13300-017-0348-2. PMC5801234. PMID29209995.^ Ramsamy K, Subramaniyan R, Patra AK (2016). "An observational Study of the Association between Androgenetic Alopecia and Size of the Prostate". International Journal of Trichology. 8 (2): 6266. doi:10.4103/0974-7753.188034. PMC4989389. PMID27601858.^ "Histology and hormonal activity in senescent thinning in males". European Hair Research Society - Abstract (Conference). Archived from the original on 2012-07-23. Retrieved 2012-07-14.^ Price, V.H. (2001). "Histology and Hormonal Activity in Senescent Thinning in Males". European Hair Research Society. Archived from the original on 21 September 2013. Retrieved 20 October 2014.^ "The Bald Truth About Hair Loss In Young Men". Stephanie Whyche, InteliHealth News Service. Aug 8, 2002. Archived from the original on February 22, 2014. Retrieved Dec 16, 2012.^ Hamilton JB (1942). "Male hormone stimulation is prerequisite and an incitant in common baldness". American Journal of Anatomy. 71 (3): 451480. doi:10.1002/aja.1000710306.^ Hamilton JB (1951). "Patterned loss of hair in man; types and incidence". Annals of the New York Academy of Sciences. 53 (3): 708728. Bibcode:1951NYASA..53..708H. doi:10.1111/j.1749-6632.1951.tb31971.x. PMID14819896. S2CID32685699.^ James, William; Berger, Timothy; Elston, Dirk (2005). Andrews' Diseases of the Skin: Clinical Dermatology. (10th ed.). Saunders. ISBN 0-7216-2921-0.^ Diagnosing Men's Hair Loss: Norwood Scale Chart Archived 2009-04-09 at the Wayback Machine. Webmd.com (2010-03-01). Retrieved on 2010-11-28.^ Rudnicka L, Olszewska M, Rakowska A, Kowalska-Oledzka E, Slowinska M (July 2008). "Trichoscopy: a new method for diagnosing hair loss". Journal of Drugs in Dermatology. 7 (7): 651654. PMID18664157.^ Mounsey AL, Reed SW (August 2009). "Diagnosing and treating hair loss". American Family Physician. 80 (4): 356362. PMID19678603.^ Yoo HG, Kim JS, Lee SR, Pyo HK, Moon HI, Lee JH, etal. (June 2006). "Perifollicular fibrosis: pathogenetic role in androgenetic alopecia". Biological & Pharmaceutical Bulletin. 29 (6): 12461250. doi:10.1248/bpb.29.1246. hdl:10371/22553. PMID16755026.^ Rashid RM, Thomas V (August 2010). "Androgenic pattern presentation of scarring and inflammatory alopecia". Journal of the European Academy of Dermatology and Venereology. 24 (8): 979980. doi:10.1111/j.1468-3083.2009.03557.x. PMID20059630. S2CID39272849.^ Khandpur S, Suman M, Reddy BS (August 2002). "Comparative efficacy of various treatment regimens for androgenetic alopecia in men". The Journal of Dermatology. 29 (8): 489498. doi:10.1111/j.1346-8138.2002.tb00314.x. PMID12227482. S2CID20886812.^ a b Darwin E, Heyes A, Hirt PA, Wikramanayake TC, Jimenez JJ (2017-12-21). "Low-level laser therapy for the treatment of androgenic alopecia: a review". Lasers in Medical Science. 33 (2). Springer Science and Business Media LLC: 425434. doi:10.1007/s10103-017-2385-5. ISSN0268-8921. PMID29270707. S2CID23783876.^ Zhou Y, Chen C, Qu Q, Zhang C, Wang J, Fan Z, etal. (2020). "The effectiveness of combination therapies for androgenetic alopecia: A systematic review and meta-analysis". Dermatologic Therapy. 33 (4). Hindawi Limited: e13741. doi:10.1111/dth.13741. ISSN1396-0296. PMID32478968.^ a b c Varothai S, Bergfeld WF (July 2014). "Androgenetic alopecia: an evidence-based treatment update". American Journal of Clinical Dermatology. 15 (3): 217230. doi:10.1007/s40257-014-0077-5. PMID24848508. S2CID31245042.^ a b c d e f Yim E, Nole KL, Tosti A (December 2014). "5-Reductase inhibitors in androgenetic alopecia". Current Opinion in Endocrinology, Diabetes, and Obesity (Review). 21 (6): 493498. doi:10.1097/MED.0000000000000112. PMID25268732. S2CID30008068.^ "Minoxidil solution". DermNet NZ. Dec 29, 2013. Archived from the original on February 2, 2014. Retrieved Jan 29, 2014.^ a b Rogers NE, Avram MR (October 2008). "Medical treatments for male and female pattern hair loss". Journal of the American Academy of Dermatology. 59 (4): 547566, quiz 5668. doi:10.1016/j.jaad.2008.07.001. PMID18793935.^ Fischer T, Hnggi G, Innocenti M, Elsner P, Treb R (2012). "Topical Melatonin for Treatment of Androgenetic Alopecia". International Journal of Trichology. 4 (4): 236245. doi:10.4103/0974-7753.111199. PMC3681103. PMID23766606.^ Hatem S, Nasr M, Moftah NH, Ragai MH, Geneidi AS, Elkheshen SA (3 October 2018). "Clinical cosmeceutical repurposing of melatonin in androgenic alopecia using nanostructured lipid carriers prepared with antioxidant oils". Expert Opinion on Drug Delivery. 15 (10): 927935. doi:10.1080/17425247.2018.1517740. PMID30169980. S2CID52139971.^ Hatem S, Nasr M, Moftah NH, Ragai MH, Geneidi AS, Elkheshen SA (15 September 2018). "Melatonin vitamin C-based nanovesicles for treatment of androgenic alopecia: Design, characterization and clinical appraisal". European Journal of Pharmaceutical Sciences. 122: 246253. doi:10.1016/j.ejps.2018.06.034. ISSN1879-0720. PMID29981403. S2CID51600289. Archived from the original on 21 September 2022. Retrieved 18 September 2022.^ English RS, Ruiz S (March 2022). "Use of Botulinum Toxin for Androgenic Alopecia: A Systematic Review". Skin Appendage Disorders. 8 (2): 93100. doi:10.1159/000518574. ISSN2296-9195. PMC8928186. PMID35415183.^ a b c van Zuuren EJ, Fedorowicz Z, Schoones J (May 2016). "Interventions for female pattern hair loss". The Cochrane Database of Systematic Reviews. 2016 (5): CD007628. doi:10.1002/14651858.CD007628.pub4. PMC6457957. PMID27225981.^ Carvalho RM, Santos LD, Ramos PM, Machado CJ, Acioly P, Frattini SC, etal. (January 2022). "Bicalutamide and the new perspectives for female pattern hair loss treatment: What dermatologists should know". Journal of Cosmetic Dermatology. 21 (10): 41714175. doi:10.1111/jocd.14773. PMID35032336. S2CID253239337.^ Nestor MS, Ablon G, Gade A, Han H, Fischer DL (December 2021). "Treatment options for androgenetic alopecia: Efficacy, side effects, compliance, financial considerations, and ethics". Journal of Cosmetic Dermatology. 20 (12): 37593781. doi:10.1111/jocd.14537. PMC9298335. PMID34741573. S2CID243801494.^ Caroli S, Pathomvanich D, Amonpattana K, Kumar A (2011). "Current status of hair restoration surgery". International Surgery. 96 (4): 345351. doi:10.9738/cc31.1. PMID22808618.^ Rose PT (August 2011). "The latest innovations in hair transplantation". Facial Plastic Surgery. 27 (4): 366377. doi:10.1055/s-0031-1283055. PMID21792780. S2CID260138453.^ Liu KH, Liu D, Chen YT, Chin SY (2019-01-31). "Comparative effectiveness of low-level laser therapy for adult androgenic alopecia: a system review and meta-analysis of randomized controlled trials". Lasers in Medical Science. 34 (6). Springer Science and Business Media LLC: 10631069. doi:10.1007/s10103-019-02723-6. ISSN0268-8921. PMID30706177. S2CID59524423.^ Gupta AK, Carviel JL (2019-11-20). "Meta-analysis of photobiomodulation for the treatment of androgenetic alopecia". Journal of Dermatological Treatment. 32 (6). Informa UK Limited: 643647. doi:10.1080/09546634.2019.1688755. ISSN0954-6634. PMID31746251. S2CID208185306.^ Zhang Y, Su J, Ma K, Fu X, Zhang C (2022-04-25). "Photobiomodulation Therapy With Different Wavebands for Hair Loss: A Systematic Review and Meta-Analysis". Dermatologic Surgery. 48 (7). Ovid Technologies (Wolters Kluwer Health): 737740. doi:10.1097/dss.0000000000003472. ISSN1076-0512. PMID35510860. S2CID248526019.^ a b Gupta AK, Bamimore MA, Foley KA (2020-04-13). "Efficacy of non-surgical treatments for androgenetic alopecia in men and women: a systematic review with network meta-analyses, and an assessment of evidence quality". Journal of Dermatological Treatment. 33 (1). Informa UK Limited: 6272. doi:10.1080/09546634.2020.1749547. ISSN0954-6634. PMID32250713. S2CID215405183. Archived from the original on 2024-02-24. Retrieved 2023-09-04.^ S L, P V, Y P, P J, T T (2021). "A Systematic Review and Meta-analysis of Randomized Controlled Trials of United States Food and Drug Administration-Approved, Home-use, Low-Level Light/Laser Therapy Devices for Pattern Hair Loss: Device Design and Technology". The Journal of Clinical and Aesthetic Dermatology. 14 (11): E64 E75. ISSN1941-2789. PMC8675345. PMID34980962.^ a b Tejapira K, Yongpisarn T, Sakpuwadol N, Suchonwanit P (2022-11-24). "Platelet-rich plasma in alopecia areata and primary cicatricial alopecias: A systematic review". Frontiers in Medicine. 9. Frontiers Media SA. doi:10.3389/fmed.2022.1058431. ISSN2296-858X. PMC9731377. PMID36507528.^ Gentile P, Garcovich S (2020-04-13). "Systematic Review of Platelet-Rich Plasma Use in Androgenetic Alopecia Compared with Minoxidil, Finasteride, and Adult Stem Cell-Based Therapy". International Journal of Molecular Sciences. 21 (8). MDPI AG: 2702. doi:10.3390/ijms21082702. ISSN1422-0067. PMC7216252. PMID32295047.^ a b Evans AG, Mwangi JM, Pope RW, Ivanic MG, Botros MA, Glassman GE, etal. (2020-05-26). "Platelet-rich plasma as a therapy for androgenic alopecia: a systematic review and meta-analysis". Journal of Dermatological Treatment. 33 (1). Informa UK Limited: 498511. doi:10.1080/09546634.2020.1770171. ISSN0954-6634. PMID32410524. S2CID218648227. Archived from the original on 2024-02-24. Retrieved 2023-09-04.^ Gupta AK, Cole J, Deutsch DP, Everts PA, Niedbalski RP, Panchaprateep R, etal. (2019). "Platelet-Rich Plasma as a Treatment for Androgenetic Alopecia". Dermatologic Surgery. 45 (10). Ovid Technologies (Wolters Kluwer Health): 12621273. doi:10.1097/dss.0000000000001894. ISSN1076-0512. PMID30882509. S2CID81980415.^ Gupta A, Bamimore M (2022-08-01). "Platelet-Rich Plasma Monotherapies for Androgenetic Alopecia: A Network Meta-Analysis and Meta-Regression Study". Journal of Drugs in Dermatology. 21 (9). SanovaWorks: 943952. doi:10.36849/jdd.6948. ISSN1545-9616. PMID36074501. S2CID252120370.^ Oth O, Stene JJ, Glineur R, Vujovic A (2018). "Injection of PRP (Platelet-rich plasma) as a treatment for androgenetic alopecia: a systematic review of the literature". Revue Mdicale de Bruxelles. 39 (5). AMUB/Revue Mdicale de Bruxelles: 438446. doi:10.30637/2018.17-056. ISSN0035-3639. PMID29869472.^ Banka N, Bunagan MJ, Shapiro J (January 2013). "Pattern hair loss in men: diagnosis and medical treatment". Dermatologic Clinics. 31 (1): 129140. doi:10.1016/j.det.2012.08.003. PMID23159182.^ a b Levy LL, Emer JJ (August 2013). "Female pattern alopecia: current perspectives". International Journal of Women's Health. 5: 541556. doi:10.2147/IJWH.S49337. PMC3769411. PMID24039457.^ a b Rogers NE, Avram MR (October 2008). "Medical treatments for male and female pattern hair loss". Journal of the American Academy of Dermatology. 59 (4): 547566, quiz 5678. doi:10.1016/j.jaad.2008.07.001. PMID18793935.^ Rondanelli M, Perna S, Peroni G, Guido D (June 2016). "A bibliometric study of scientific literature in Scopus on botanicals for treatment of androgenetic alopecia". Journal of Cosmetic Dermatology. 15 (2): 120130. doi:10.1111/jocd.12198. PMID26608588. S2CID20224420.^ Prager N, Bickett K, French N, Marcovici G (April 2002). "A randomized, double-blind, placebo-controlled trial to determine the effectiveness of botanically derived inhibitors of 5-alpha-reductase in the treatment of androgenetic alopecia". Journal of Alternative and Complementary Medicine. 8 (2): 143152. doi:10.1089/107555302317371433. PMID12006122. S2CID5866411.^ "Baldness Breakthrough: microRNA Stimulates Hair Growth in Aging Follicles". SciTechDaily. 2023-06-08. Archived from the original on 2023-06-09. Retrieved 2023-06-09.^ Cash TF (September 1999). "The psychosocial consequences of androgenetic alopecia: a review of the research literature". The British Journal of Dermatology. 141 (3): 398405. doi:10.1046/j.1365-2133.1999.03030.x. PMID10583042. S2CID40583229.^ Cash TF (June 1992). "The psychological effects of androgenetic alopecia in men". Journal of the American Academy of Dermatology. 26 (6): 926931. doi:10.1016/0190-9622(92)70134-2. PMID1607410.^ Birch MP, Lalla SC, Messenger AG (July 2002). "Female pattern hair loss". Clinical and Experimental Dermatology. 27 (5): 383388. doi:10.1046/j.1365-2230.2002.01085.x. PMID12190638. S2CID39467830.^ "Women and Hair Loss: The Causes". Archived from the original on 30 June 2010. Retrieved 2010-06-29.^ Cranwell W, Sinclair R, Feingold KR, Anawalt B, Boyce A, Chrousos G, etal. (2000). "Male Androgenetic Alopecia". Endotext. MDText.com. PMID25905192. Archived from the original on 2022-05-28. Retrieved 2022-06-29.^ Thomas TL, Patel GL (1976). "Optimal conditions and specificity of interaction of a distinct class of nonhistone chromosomal proteins with DNA". Biochemistry. 15 (7): 14811489. doi:10.1021/bi00652a019. PMID4089. Archived from the original on 2024-10-10. Retrieved 2022-06-30.^ Henss R (2001). "Social Perceptions of Male Pattern Baldness. A Review". Dermatology and Psychosomatics. 2 (2): 6371. doi:10.1159/000049641. S2CID143994546.^ Castle S (2002). "IT LOCKS LIKE GIRLS GO FOR DARKER HAIR; Bald men sexy too says survey". The Free Library. Archived from the original on 2015-05-18. Retrieved 2015-05-14.^ Kabai P (August 2010). "Might early baldness protect from prostate cancer by increasing skin exposure to ultraviolet radiation?". Cancer Epidemiology. 34 (4): 507. doi:10.1016/j.canep.2010.04.014. PMID20451486.^ Lotufo PA, Chae CU, Ajani UA, Hennekens CH, Manson JE (January 2000). "Male pattern baldness and coronary heart disease: the Physicians' Health Study". Archives of Internal Medicine. 160 (2): 165171. doi:10.1001/archinte.160.2.165. PMID10647754.^ Gatherwright J, Amirlak B, Rowe D, Liu M, Gliniak C, Totonchi A, etal. (2011). "The Relative Contribution of Endogenous and Exogenous Factors to Male Alopecia". Plastic and Reconstructive Surgery. 128: 14. doi:10.1097/01.prs.0000406222.54557.75. S2CID74776022.^ Schmidt JB (1994). "Hormonal basis of male and female androgenic alopecia: clinical relevance". Skin Pharmacology. 7 (12): 6166. doi:10.1159/000211275. PMID8003325.^ Remes K, Kuoppasalmi K, Adlercreutz H (June 1985). "Effect of physical exercise and sleep deprivation on plasma androgen levels: modifying effect of physical fitness". International Journal of Sports Medicine. 6 (3): 131135. doi:10.1055/s-2008-1025825. PMID4040893. S2CID25897282.^ Toone BK, Wheeler M, Nanjee M, Fenwick P, Grant R (September 1983). "Sex hormones, sexual activity and plasma anticonvulsant levels in male epileptics". Journal of Neurology, Neurosurgery, and Psychiatry. 46 (9): 824826. doi:10.1136/jnnp.46.9.824. PMC1027564. PMID6413659.^ Davidson JM, Kwan M, Greenleaf WJ (November 1982). "Hormonal replacement and sexuality in men". Clinics in Endocrinology and Metabolism. 11 (3): 599623. doi:10.1016/S0300-595X(82)80003-0. PMID6814798.^ Crabtree JS, Kilbourne EJ, Peano BJ, Chippari S, Kenney T, McNally C, etal. (May 2010). "A mouse model of androgenetic alopecia". Endocrinology. 151 (5): 23732380. doi:10.1210/en.2009-1474. PMID20233794.^ Sundberg JP, King LE, Bascom C (2001). "Animal models for male pattern (androgenetic) alopecia". European Journal of Dermatology. 11 (4): 321325. PMID11399538.^ Sundberg JP, Beamer WG, Uno H, Van Neste D, King LE (October 1999). "Androgenetic alopecia: in vivo models". Experimental and Molecular Pathology. 67 (2): 118130. doi:10.1006/exmp.1999.2276. PMID10527763.^ Borzo G (2002). "Unique social system found in famous Tsavo lions". EurekAlert. Archived from the original on 2014-02-22. Retrieved 2013-09-21.Wikimedia Commons has media related to Androgenic alopecia.NLM- Genetics Home ReferenceScow DT, Nolte RS, Shaughnessy AF (April 1999). "Medical treatments for balding in men". American Family Physician. 59 (8): 218994, 2196. PMID10221304.Retrieved from " As a library, NLM provides access to scientific literature. Inclusion in an NLM database does not imply endorsement of, or agreement with, the contents by NLM or the National Institutes of Health. Learn more: PMC Disclaimer | PMC Copyright Notice Female pattern hair loss (FPHL) is the most common form of alopecia in women. Affected women may experience psychological distress and impaired social functioning. Early diagnosis and initiation of treatment are desirable because treatments are more effective to avoid the progression of hair loss than stimulating regrowth. Typically, a diagnosis of FPHL can be confirmed by review of a patient's medical history and a physical examination alone. Testing a scalp biopsy is diagnostic but usually not required. In women with signs of hyperandrogenism, an investigation for ovarian or adrenal disorders should be performed. Treatment for FPHL is obscured by myths. The aim of FPHL treatment could be two-fold: Reverse or stabilize the process of hair follicle miniaturization. Mild-to-moderate FPHL in women can be treated with oral antiandrogen therapies (cyproterone acetate and spironolactone) and/or topical minoxidil with good results in many cases. If used correctly, available medical treatments arrest the progression of the disease and reverse miniaturization in most patients with mild-to-moderate FPHL. Hair systems and surgery may be considered for selected cases of severe FPHL.Keywords: Androgenetic alopecia, female pattern hair loss, finasteride, minoxidil, platelet-rich plasmaFemale pattern hair loss (FPHL) has emerged as the preferred term for androgenetic alopecia (AGA) in women due to the uncertain relationship between androgens and this entity (Olsen, 2001). FPHL is the most common hair loss disorder in women. Initial symptoms may develop during the teenage years and lead to progressive hair loss with a characteristic pattern distribution (Vujovic and Del Marmol, 2014). FPHL is characterized as a nonscarring diffuse alopecia that evolves from the progressive miniaturization of hair follicles and subsequent reduction in the number of hairs, especially in the central, frontal, and parietal scalp regions (Olsen, 2002; Fig. 1). Clinical example of female pattern hair lossFPHL has three main clinical manifestations. The first manifestation is the diffuse thinning of the upper biparietal and vertex regions and preservation of the anterior hair implantation line. There are several hair loss scales that attempt to categorize FPHL and each has advantages and disadvantages (Ludwig, 1977, Ramos and Miot, 2015, Savin, 1994, Yip and Sinclair, 2006; Fig. 2, Fig. 3). A wide discussion of each scale is not the primary scope of the paper. Ludwig scale representation Sinclair Scale Sinclairs classification. MPA is divided into four levels of intensity on the basis of normal scalp to the left (Sinclair et al., 2005).Another manifestation is the thinning of the upper bitemporal region and vertex with frontal accentuation that configures as a triangular or Christmas tree form with hair loss in a triangular shape in the frontal-vertical area (Olsen, 1999; Fig. 4). A third manifestation is a deep recession of the frontal-temporal hairline and true vertex balding, which is typically seen in men but occasionally occurs in women although uncommon (Redler et al., 2017). Olsens classification. Olsen patterns incorporate the accentuation of the front-overtical alopecia, which has a triangular or Christmas tree form with hair loss in a triangular form in the front-overtical area (Olsen, 2002).Several management options are available to treat FPHL but every treatment usually requires a long period of time to get a significative improvement. Therefore, a considerable amount of time should always be dedicated to doctor-patient counseling to improve compliance.The frequency of FPHL varies among population groups and ordinarily increases with age. However, a comparison of prevalence between different studies is hampered by the lack of universally accepted criteria for the disease (Ramos and Miot, 2015). Among healthy women, approximately 6% to 38% experience some degree of frontal and/or frontal-parietal hair loss (Birch et al., 2001).The age of onset for FPHL is during the reproductive years, which is later than in men. Twelve percent of women first develop clinically detectable FPHL by age 29 years, 25% by age 49 years, 41% by 69 years, and >50% have some element of FPHL by 79 years (Birch et al., 2002). More severe cases of the disease during puberty are more rarely described. Nevertheless, there is a greater demand for treatment among patients ages 25 to 40 years (Tosti and Piraccini, 2006). In the United Kingdom, 6% of women younger than age 30 years have FPHL for women older than 70 years, FPHL reaches a rate of 42% (Birch et al., 2002). Only 43% of women age >80 years show no evidence of FPHL (Sinclair and Dawber, 2001).FPHL and male AGA share a final common pathway that causes follicular regression but current knowledge suggests that the etiology is not necessarily the same in both sexes. Although the role of androgens in the pathogenesis of male hair loss has been clearly established, the role of androgens in FPHL is less clear. In fact, FPHL may develop even in the absence of androgens (Herskovitz and Tosti, 2013). However, it is likely that other nonandrogenic factors that are currently unidentified may play a role in the pathogenesis of FPHL (Redler et al., 2017). Therefore, the involvement of these genes in the etiopathogenesis of FPHL cannot be completely excluded.In women with FPHL who do not have elevated androgen levels, a genetic predisposition may be involved. This genetic disposition permits normal levels of circulating androgen to act on follicular target cells, which are specially sensitized by binding to specific intracellular androgen receptors. In other cases, an androgen-independent mechanism may be involved in the development of FPHL (Orme et al., 1999). Two recent studies by Heilmann-Heimbach et al. (2017) and Pickrell et al. (2016) have substantially found an increased number of gene loci (>60) associated with male AGA.Hair loss in women is polygenic and multifactorial with the additional influence of environmental factors. Several studies focused on the importance of several genes related to alopecia (Carey et al., 1993, Hillmer et al., 2008, Randall, 2008). FPHL involves progressive hair follicle miniaturization and subsequently the conversion of terminal follicles into vellus-like follicles. These vellus-like follicles have a shortened hair cycle because of a reduction in the anagen phase, which leads to the production of short and fine hair shafts. Unlike in men, the miniaturization is not uniform and intense in women; therefore, there are no complete areas of baldness except in very rare cases (Birch et al., 2001). Moreover, the miniaturization process may be accompanied by a mild-to-moderate lympho-histiocytic inflammatory infiltrate in the peri-infundibular region. The term microinflamation has been used to differentiate this infiltrate from the inflammation that occurs in scarring alopecia (Stefanato, 2010).FPHL and male balding share a final common pathway of follicular regression but current knowledge suggests that the etiology is not necessarily the same in both sexes. Androgens are a key driver of male balding and also involved in the etiology of pattern hair loss in some women. However, other nonandrogenic factors that are still unidentified likely play a role in causing FPHL (Herskovitz and Tosti, 2013).The most common endocrinologic comorbidity that is associated with FPHL is polycystic ovarian syndrome (El Sayed et al., 2016). Metabolic syndrome, which is characterized by obesity, insulin resistance, hypertension, hyperprolactinemia, and raised aldosterone levels, also appears to be frequently associated with FPHL (El Sayed et al., 2016). An increased risk of carotid and coronary artery diseases have also been reported (Arias-Santiago et al., 2010). To further clarify the comorbidity profile of FPHL, systematic studies in larger population-based samples are needed.An association between ferritin levels and FPHL is controversial. Some studies have demonstrated lower ferritin levels in patients with FPHL compared with controls and antiandrogen therapy seem to work better in patients with ferritin levels >40 g/l (Ramos and Miot, 2015).Women with increased hair shedding but little or no reduction in hair volume over the mid-frontal scalp could be suffering from several diseases and acute and chronic telogen effluvium (TE) should be considered in particular. Anamnesis and a physical examination are needed to get the right diagnosis. Anamnesis should focus on when the hair loss started, whether the loss was gradual or involved handfuls of hair as well as any physical, mental, or emotional stressors that may have occurred within the previous 3 to 6 months. A history and physical examination should aim at detecting signs of hyperandrogenism such as hirsutism, ovarian abnormalities, menstrual irregularities, acne, and infertility. Laboratory test results are rarely evaluated in women who suffer from FPHL with no signs of hyperandrogenism.Moreover, hair loss may occur in patients who are treated with oral contraceptive medications that contain progesterone with a high androgenic potential such as norethindrone or who recently discontinued an estrogenic oral contraceptive medication that was taken for a long period of time. A physical examination should include all aspects of the scalp and especially evaluate the involvement of the occipital area, which will show a widening of the central part with a diffuse reduction in hair density over the frontal scalp rather than baldness per se. Although these areas show the most marked reduction in hair density, evidence of global reduction in hair density usually exists throughout the all scalp.The pull test is an evaluation of the number of hairs that are shed after a slight traction on the scalp hair. This test helps to roughly estimate the severity of hair loss in daily practice with a high interobserver variability. A bundle of approximately 50 to 60 hairs is grasped between the thumb, index finger, and middle finger from the base close to the scalp. The hair is firmly but not forcibly tugged away from the scalp as the fingers slide along the hair shaft. Afterward, the number of extracted hairs is counted. If more than 10% of the grasped hair (six hairs) are pulled away from the scalp, the pull test is positive and implies active hair shedding. Fewer than six hairs that are easily pulled out is considered normal physiologic shedding.Nonetheless, McDonald et al. (2017) recently tried to quantify normal hair pull test values by elucidating the effect of pretest hair washing and brushing and performing the pull test on 181 participants. The study showed that normal values for the hair pull test should be reduced to 2 hairs or fewer. In addition, neither hair brushing nor washing altered the hair pull test results. Hair washing and brushing may now occur at any time before the hair pull test instead of 4 to 5 days prior. The pull test is only a rough approach to diagnosis and more reliable tests and objective measurements should be added.With the standardized wash test, women refrain from shampooing for 5 days and then shampoo and rinse their hair in a basin with the hole covered with gauze. All the hairs that remain in the water and gauze are collected and sent for examination. A total of 34 hairs must be counted and divided into 3 cm and 5 cm in length. This is an important technique to differentiate TE from FPHL.The wash test was modified by Rebora et al. (2005) and called the AGA/TE wash test to distinguish between AGA and TE. For the AGA/TE modified wash test, the hairs are counted and divided into three groups by hair length: 1) Long hair >5 cm, 2) intermediate-length hair (>3 to 20% is diagnostic for AGA), short vellus hair (7 vellus hairs on the frontal scalp), yellow dots (sign of severe miniaturization and more numerous in patients with severe FPHL), pinpoint white dots and scalp pigmentation (honeycombed-like pattern on sun-exposed scalp), peripilar sign (subtle brown halo, which is a specific finding in early stages of the disease and reflects perifollicular inflammation; focal areas of baldness [atrichia] could be recognized in postmenopausal women), and scalp biopsy (best way to distinguish between chronic TE and FPHL [Sinclair et al., 2004] through the calculation of the terminal-to-vellus hair ratio; ratio of 8:1 is considered diagnostic of chornic TE). Scalp biopsy is an invasive technique and the indication for a scalp biopsy should be carefully provided but, when necessary, the intervention should not be delayed.A differential diagnosis of FPHL includes TE, postpartum hair loss, cicatricial alopecia in pattern distribution, and alopecia areata (diffuse or incognita; Asz-Sigall et al., 2016). As discussed, dermoscopy is a very useful complementary tool to get the right diagnosis, especially in the early stages of the disease.Since FPHL can mimic and often runs concurrently with other diagnoses, a detailed medical history overview and physical examination should be performed on the patient. With the help of other diagnostic tools and techniques, other concurrent conditions should be investigated and treated if applicable.Treatment options available for FPHL can be classified in two categories: Topical and systemic drugs. Because FPHL is a biological process determined by a sensitivity to androgens that are genetically mediated, most of these drugs act on the androgen activity by altering the production, transport, or metabolism of androgens or preventing the binding to androgenic receptors. Moreover, androgen-dependent medications may cause abnormalities in the genitalia of the male fetus; thus, these drugs are contraindicated in pregnant women. This finding leads many physicians to recommend an oral contraceptive therapy throughout the entire course of treatment.Minoxidil is a piperidinopyrimidine derivative and potent vasodilator that is effective orally for severe hypertension. The drug was approved in 1979 by the U.S. Food and Drug Administration (FDA) for the treatment of hypertension. Minoxidil was first noticed to improve hair loss in male AGA in 1980 when used topically (Varothai and Bergfeld, 2014). Minoxidil solutions of 2% and 5% were approved for the treatment of male AGA in 1988 and 1991, respectively. In FPHL, 2% minoxidil was approved by the FDA in 1991 and a 5% minoxidil foam with once daily application was approved in 2014 (Varothai and Bergfeld, 2014).Minoxidil is effective in both sexes (Blumeyer et al., 2011, van Zuuren et al., 2012a, van Zuuren et al., 2012b) and statistically significantly increases nonvellus and total hair count at 24 weeks of treatment. In male AGA, the 5% solution shows better results than the 2% solution (Tsuboi et al., 2009). For FPHL, no study has compared the effectiveness of the 2% minoxidil solution twice daily with the 5% minoxidil solution once daily but a randomized trial revealed that the 5% minoxidil foam once daily was similar in effectiveness to the 2% minoxidil solution twice daily for the treatment of female AGA (Blume-Peytavi et al., 2011).Minoxidil is a potassium channel opener and stimulates hair growth by increasing the anagen phase of the hair cycle. Minoxidil enhances angiogenesis around the follicle but the exact mechanisms are currently unknown (Blumeyer et al., 2011, Gupta and Foley, 2014). An activation of cyto-protective prostaglandin synthase-1 may exist that increases hair count and weight. The topical minoxidil 2% solution should be applied only to the affected area of the scalp at 1 ml twice daily (once daily for the minoxidil 5% foam) for a minimum period of 12 months before determining the efficacy. The clinical response to 5% topical minoxidil for the treatment of AGA is typically observed after 3 to 6 months and approximately 40% of patients show a significative improvement. For this reason, minoxidil response testing to rule out nonresponders has significant clinical utility.When effective, treatment should be continued indefinitely as with a chronic disease because discontinuation may induce TE in the minoxidil-dependent hair within 4 to 6 months (Banka et al., 2013). Patients should also be warned that during the first months of treatment, a transient increase shedding may occur. Treatment side effects are uncommon and include allergic or irritative contact dermatitis, which is more commonly related to the solution vehicle propylene glycol. This can be overcome with use of the 5% foam that does not contain this ingredient. Another possible side effect is hypertrichosis of the forehead or face, which is usually caused by accidental contamination or improper application (Herskovitz and Tosti, 2013). Additionally, the 5% minoxidil foam provides an alternative option for women who do not wish or are unable to use oral anti-androgen or hormonal contraceptive medications (Gupta and Foley, 2014).Minoxidil is a pro-drug that is converted to its active form, minoxidil sulfate, by sulfotransferase enzymes in the outer root sheath of hair. Minoxidil sulfate is the active form that is required for both the promotion of hair regrowth and the vasodilatory effects of minoxidil. Several studies have demonstrated that sulfotransferase enzyme activity in plucked hair follicles predicts topical minoxidil response in patients with FPHL. Moreover, different studies have been conducted to confirm the clinical utility of a sulfotransferase activity assay to guide treatment (Goren et al., 2015). These analysis show that the sulfotransferase enzyme test can successfully rule out 95.9% of nonresponders to topical minoxidil for the treatment of AGA (McCoy et al., 2016).Latanoprost and bimatoprost were initially developed for eye glaucoma when the growth of eye lashes was noticed as a side effect. In fact, among the prostaglandins (PG), the PG-F2 analog treatments latanoprost and bimatoprost are known to stimulate hair growth by prolonging the anagen phase (Valente Duarte de Sousa and Tosti, 2013). A small placebo-controlled trial in men with mild AGA showed that 0.1 % latanoprost significantly increased hair density and pigmentation at 24 weeks compared with baseline and compared with the placebo-treated site. Nevertheless, the study included only 16 male patients and the medication was applied to a very small area of the scalp (Blume-Peytavi et al., 2012, Herskovitz and Tosti, 2013). However, a case report of postmenopausal patients with FPHL failed to demonstrate the efficacy of locally injected 0.03 % bimatoprost for 16 weeks (Emer et al., 2011).Others studies have revealed that an increased PG-D2 level is correlated with the miniaturization of hair follicles and, moreover, the topical application of PG-D2 also inhibited hair growth (Garza et al., 2012). Recent research studies are looking for other drugs that are able to block the PG-D2 receptor (GPR44), which has an inhibitory effect on hair growth and is known to be elevated in the scalp of patients with AGA (Nieves and Garza, 2014). Setipiprant (KITH-105) is an orally administered GPR44 receptor inhibitor in a clinical trial for asthma and could have a potential application for patients with AGA (Keaney, 2015). A phase 2 clinical trial is evaluating the use of oral setipiprant in comparison with placebo and finasteride 1 mg/d in men ages 18 to 41 years with AGA (Study NCT02781311).The pathobiology of AGA is not completely clear. The genetic predisposition and influence of androgens have shown to play a role in AGA. However, these factors do not explain the presence of a substantial lymphoid infiltrate that abuts on the infra-infundibulum and isthmus of transitional hair follicles (Pirard et al., 1996). The negative influence of inflammation on AGA hair status is confirmed by AGA exacerbation after intercurrent episodes of other inflammatory dermatoses and especially seborrheic dermatitis.Ketoconazole (KCZ) is considered highly effective to treat dandruff and seborrheic dermatitis. KCZ has an anti-inflammatory property and also acts as androgen-receptor antagonist. These issues may explain the efficacy of topical KCZ (Pirard-Franchimont et al., 1998). The inflammation that abuts on the AGA hair follicles might also be related to the presence of some members of that microflora that normally marks seborrheic dermatitis. Hence, KCZ, by reducing AGA inflammation, improves the hair status (Pirard et al., 1996). In FPHL with hyperandrogenism, 2% ketoconazole shampoo has shown a benefit in treatment (Sonino et al., 1990).Melatonin is a pineal gland neurohormone that is released with a circadian rhythm and regulates different physiological processes such as seasonal biorhythms and daily sleep-wake cycles that influence the aging process. Melatonin is notable for its protective and anti-apoptotic effects due to its strong anti-oxidant properties and ability to actively capture free radicals (Fischer et al., 2001).Hair follicle is a target organ for numerous neurohormones, neuropeptides, neurotrophins, and neurotransmitters but also produces many of these molecules (Arck et al., 2006). Therefore, the pilosebaceous unit is best recognized as a neuroendocrine organ. In this context, melatonin modulates hair growth, pigmentation, and molting in many species including humans (Fischer et al., 2008, Singh and Jadhav, 2014). The topical application of the melatonin 0.1 % solution was shown to significantly increase anagen hair in male and female AGA with a good compliance in a controlled study (Fischer et al., 2012).Platelet-rich plasma (PRP) is an autologous concentration of human platelets contained in a small volume of plasma. Platelets can be likened to cell reservoirs that produce, store, and release numerous growth factors capable of stimulating the proliferation of stem cells and the replication of mesenchymal cells, fibroblasts, osteoblasts, and endothelial cells. PRP is composed by several different growth factors: Platelet-derived growth factor, transforming growth factor a, vascular endothelial growth factor, insulin-like growth factor 1, epidermal growth factor, basic fibroblast growth factor, transforming growth factor-b1, and platelet-activating factor that are released through degranulation and stimulate bone and soft tissue healing.The secretion of these growth factors begins within 10 minutes after clotting and >95% of the presynthesized growth factors are secreted within 1 hour. The addition of thrombin and calcium chloride actives platelets in PRP and induces the release of factors from alpha granules. In dermatology and aesthetic medicine, indications range from hair restoration such as nonsurgical therapeutic options for patients with hair loss to chronic ulcers (Dhurat and Sukesh, 2014, Fabi and Sundaram, 2014).Some hair transplant surgeons have used PRP in hair transplantation procedures, either by storing the grafts in PRP until they are placed on the scalp or by injecting PRP into the scalp prior to the placement of grafts (Rose, 2011). Although studies may differ in methodology, patient selection, and treatment technique, some authors have reported regrowth rates after five local treatments of 3 mL of PRP at 2- to 3-week intervals and histologic examinations showed thickened epithelium, proliferation of collagen fibers and fibroblasts, and increased vessels around follicles (Cervantes et al., 2018, Lee et al., 2015, Leo et al., 2015, Miao et al., 2013, Takikawa et al., 2011)-63).Microneedling is a minimally invasive dermatologic procedure in which fine needles are rolled over the skin to puncture the stratum corneum. Through the physical trauma from needle penetration, microneedling induces a wound healing cascade with minimal damage to the epidermis that induces collagen formation, neovascularization, and growth factor production of the treated areas. Microneedling has shown promising results as an adjuvant therapy for enhanced drug delivery in the treatment of atrophic scars, AGA, alopecia areata, and pigmentation disorders such as melasma.Although here are only a limited number of studies that have examined this therapy in the use of hair loss, microneedling has been successfully paired with other hair-growth promoting therapies such as minoxidil, platelet-rich plasma, and topical steroidal medications. Microneedling penetration of such first-line medications may be facilitated and is one mechanism that promotes hair growth (Fertig et al., 2018). To date, microneedling treatment has achieved the best results in AGA. Indeed, microneedling shows some promise in improving hair growth and especially in combination with existing techniques (Shah et al., 2017).Examples of combination therapies in patients with AGA (male and female) are listed in Figure 5. Summary of evidence for the use of combination treatment of topical minoxidil in male and female androgenetic alopecia (Varothai and Bergfeld, 2014)Low-level light therapy (LLLT) is a relatively new technique in the treatment of AGA. The biochemical mechanisms are not completely understood but the cellular respiratory chain of mitochondria probably absorb the light energy, which results in increased electron transport and the promotion of cellular signaling and in turn allows for hair regrowth (Rangwala and Rashid, 2012). Currently, several LLLT devices are available for the treatment of alopecia including a comb, hood, and helmet.Many research studies started investigations into the efficacy of LLLT for AGA in men and women but the power of the device together with no standardization of study methods resulted in a high risk of bias. Therefore, the efficacy of LLLT devices remains unclear. The devices that were studied the most include the Hair Max LaserComb (Lexington International, LLC, Boca Raton, FL), which a hand-held, noninvasive device with a wavelength of 655 nm that was approved by the FDA for the safe treatment of male and female AGA. Its effectiveness has been shown to statistically significantly increase terminal hair density in comparison with a control group at 6.5 months (Jimenez et al., 2014, Leavitt et al., 2009). Therefore, LLLT and particularly a 650 to 900 nm wavelength at 5 mW may be a therapeutic option for patients with AGA.Finasteride works by inhibiting the 5-reductase II enzyme, which is responsible to catalyze the conversion of testosterone to the much more active chemical 5 dihydrotestosterone. Finasteride is not FDA-approved for use in women and contraindicated in pregnant women and during lactation because of the risk of feminization of the male fetus. Large scale studies on its efficacy are currently limited. Price et al. (2000) proved the ineffectiveness of finasteride 1 mg/day taken for 12 months in postmenopausal women with AGA. However, case reports and series have demonstrated the efficacy of low-dosage finasteride in some cases of FPHL in both pre- and postmenopausal women. Shum et al. (2002) found that finasteride 1.25 mg/day improves pattern hair loss in women with hyperandrogenism but not in postmenopausal women with FPHL without hyperandrogenism. Thai and Sinclair (2002) reported on the improvement with finasteride 5 mg weekly in a woman who was intolerant to other antiandrogen therapies.Higher doses (2.5-5 mg/day) appear to be necessary to treat FPHL effectively (Iorizzo et al., 2006). In addition, Finasteride 0.05% in a gel formulation has been used for the treatment of pattern hair loss and shown promising results (Hajheydari et al., 2009). Further randomized controlled trials with finasteride (different dosages) are needed to determine optimal dosing regimens and its efficacy in FPHL.Dutasteride is a 5-reductase type I and II inhibitor that has not currently been approved in men and women for the treatment of hair loss. However, dutasteride has been approved for the treatment of benign prostatic hyperplasia at the dose of 0.5 mg daily. Dutasteride can reduce serum dihydrotestosterone levels by >90% and has been used with success at a dose of 0.5 mg daily in male AGA (Jung et al., 2014). Dutasteride has been reported to treat FPHL successfully with no side effects at doses that range from 0.25 to 0.5 mg/day (Olsen et al., 2006).Dutasteride should not be given to women of childbearing age unless they are using birth control measures because of the potential feminizing effects on the male fetus or to female patients who have test results that show impaired liver function. In another study, 25 postmenopausal women with female AGA of a male pattern were treated with dutasteride 0.25 mg/d. The results demonstrated a wide improvement that started in the frontotemporal region, followed by the vertex and frontal areas in 60% of cases at 1 year of treatment and in 80% of cases at 2 years (Camacho and Tosti,

2005).Cyproterone acetate inhibits gonadotropin-releasing hormones and blocks androgen receptors but is not available in the United States. The best study to investigate cyproterone actetate was a 12-month randomized trial that compared the use of topical minoxidil 2% and cyproterone acetate in 66 women with FPHL. The authors found that minoxidil 2% was more effective in women without evidence of hyperandrogenism but cyproterone acetate was more effective in women with multiple symptoms (Vexiau et al., 2002).In contrast, there is only one significant randomized study that has shown no benefit for cyproterone acetate (Carmina and Lobo, 2003). The treatment doses that were utilized vary but one of the most effective doses appears to be 100 mg/day on days 5 to 15 of the menstrual cycle and supplemented by 50 g ethinyl estradiol on days 5 to 25 (Dawber et al., 1982). However, there is insufficient evidence to date that oral hormonal treatment prevents progression or improves AGA in female patients. Nevertheless, a subgroup analysis suggests that oral cyproterone acetate may improve AGA in female patients with hyperandrogenism (Blumeyer et al., 2011).Spironolactone is the most commonly used, off-label anti-androgen for the treatment of female AGA and hirsutism. Spironolactone is a potassium-sparing diuretic and structural antagonist of aldosterone (van Zuuren et al., 2012a) and acts as an androgen antagonist by competitively blocking androgen receptors as well as inhibiting ovarian androgen production. The usual daily dose is 100 to 200 mg. The side effects of spironolactone are due partly to its additional actions as it may act as an aldosterone antagonist and cause postural hypotension, electrolyte disturbances, menstrual irregularities, fatigue, urticaria, breast tenderness, and hematologic disturbances. Because of these known side effects, blood pressure and electrolyte balance, especially in patients with comorbid conditions or concurrent potentially interactive medications, should be checked during the first few months of treatment.Published studies that support the efficacy of spironolactone are limited. An open intervention study concluded that spironolactone 200 mg/day was equally effective in either restoring hair growth or preventing further progression of hair loss compared with cyproterone acetate at a dose of either 50 mg/day or 100 mg/day for 10 days every menstrual cycle (Sinclair et al., 2005).Oral minoxidil is not often used in the treatment of AGA and FPHL, mainly because of the side-effect profile at standard doses. Off-label use of oral minoxidil is known to improve hair density in the treated patients but could be complicated by postural hypotension, fluid retention, and hypertrichosis. Fuid retention can often be managed by the addition of spironolactone but has the potential to increase postural hypotension. As minoxidil side effects are all dose-related, the dose could be 0.25 mg every day (Sinclair, 2018). Low-dose oral minoxidil is usually well tolerated in the majority of patients with FPHL and a reasonable alternative in women who are intolerant of or unwilling to use topical minoxidil (Sinclair, 2018).The benefit of oral supplementation with amino acids, biotin, zinc, and other micronutrients in hair loss of any origin is controversial. For patients with TE, oral supplementation with L-cystine and B-complex vitamins showed a normalization of the anagen hair rate in a TrichoScan analysis but the effectiveness for FPHL is doubtful (Singal et al., 2013).Rasheed et al. (2013) examined the relation between insufficiency/deficiency of serum ferritin and vitamin D and between TE or FPHL and demonstrated relation in both conditions. There are several mechanisms by which both iron and vitamin D have possible effects on hair growth. As the role of iron and ferritin levels increase in nondividing cells, rapidly proliferating cells such as hair follicle matrix cells have lower levels of ferritin and higher levels of free iron. This balance of ferritin and iron is partially controlled by the transcription factor c-Myc (Rasheed et al., 2013). Overexpression of c-Myc in the cutaneous epithelium results in a loss of follicular differentiation and decrease in stem cells but whether this phenotype is related to an abnormal iron metabolism remains to be determined.Furthermore, iron acts as a metabolic cofactor for ribonucleotide reductase, which is the rate-limiting enzyme for DNA synthesis of hair growth stems. Therefore, the depletion of iron results in the inhibition of proliferation. Vitamin D has been suggested as an optimal concentration of this micronutrient that is necessary to delay aging phenomena including hair loss. Data from animal models show that vitamin D receptor activation plays an important role in anagen initiation and recent data suggested that vitamin D receptors regulate the expression of genes that are required for hair follicle cycling (Amor et al., 2010, Demay, 2012). Also, the definition of iron deficiency in hair loss remains an important question because some studies suggest that with a serum ferritin level of 30 g/l and active hair loss, patients should be treated with iron therapy. However, other studies suggest higher cutoff limits such as 40 g/l and 70 g/l or lower cutoff limits such as 10 g/l to 15 g/l (Bregy and Trueb, 2008, Deloche et al., 2007, Rushton and Ramsay, 1992).Other ingredients such as saw palmetto (Nutrafol) or marine protein complexes (Viviscal) may have anti-androgenic and anti-inflammatory properties but their efficacy alone as a monotherapy needs more investigation.Hair transplantation is an important option for patients over 25 years of age with FPHL who do not have success with medical therapies when the hair loss has been stabilized (Atanaskova Mesinkovska and Bergfeld, 2013). Hair transplantation involves the relocation or transfer of hairs from the occipital to the bald area. Ideal surgical candidates for hair transplantation are women with high hair density in the donor site over the occipital scalp and extensive hair loss or thinning of the frontal scalp.Follicular unit hair transplantation is a surgical treatment of baldness in which follicular units of hair (consisting of naturally occurring bundles of hairs) are dissected under a stereomicroscope and transplanted in the bald area to give a natural look. The procedure is performed under local anesthesia whereby one session involves the transplantation of 800 to 1200 grafts. Thanks to the use of follicular unit transplantation, the technique has become much less invasive and produces natural, undetectable, and reproducible results.The most common problems that are encountered in hair transplantation in women are related to insufficient hair donor areas, the need for magnification to insert the grafts between the existing hair follicles in the recipient area, and temporary worsening of global aspect after the transplant. To achieve a good result, the correct selection of ideal candidates is important for this procedure. A new trend in hair transplantations is the adjuvant use of PRP. The growth factors and plasma components can be injected directly into the scalp before placement of the grafts or the hair grafts may be stored in PRP until placed on the scalp (Carter et al., 2011, Rose, 2011). Moreover, robotic systems can select and remove individual hair follicles from the donor area with great precision and without fatigue (Pereira et al., 2016).Despite the high prevalence of FPHL, its management still imposes several difficulties to dermatologists' clinical practice. The investigation must be improved to identify the potential elements (other than genetic and hormonal) that are involved in the pathogenesis of FPHL. These findings are needed to develop new and more effective therapies to prevent and reverse the diseases course. Since the response to treatment of FPHL is a challenge for dermatologists, new data with regard to the epidemiology, genetics, and pathophysiology of FPHL may help to improve the quality of life of patients affected by this disease.Amor K.T., Rashid R.M., Mirmirani P. Does D matter? The role of vitamin D in hair disorders and hair follicle cycling. Dermatol Online J. 2010;16:3. [PubMed] [Google Scholar]Arck P.C., Slominski A., Theoharides T.C., Peters E.M.J., Paus R. Neuroimmunology of stress: Skin takes center stage. J Invest Dermatol. 2006;126:16971704. doi: 10.1038/sj.jid.5700104. [DOI] [PMC free article] [PubMed] [Google Scholar]Arias-Santiago S., Gutirrez-Salmern M.T., Castellote-Caballero L., Buenda-Eisman A., Naranjo-Sintes R. Androgenetic alopecia and cardiovascular risk factors in men and women: A comparative study. J Am Acad Dermatol. 2010;63:420429. doi: 10.1016/j.jaad.2009.10.018. [DOI] [PubMed] [Google Scholar]Asz-Sigall D., Gonzlez-de-Cossio-Hernndez A.C., Rodrguez-Lobato E., Ortega-Springall M.F., Vega-Memije M.E., GuzmnSkin R.A. Differential diagnosis of female-pattern hair loss. Skin Appendage Disord. 2016;2:1821. doi: 10.1159/000445806. [DOI] [PMC free article] [PubMed] [Google Scholar]Atanaskova Mesinkovska N., Bergfeld W.F. Hair: What is new in diagnosis and management? Female pattern hair loss update: diagnosis and treatment. Dermatol Clin. 2013;31:119127. doi: 10.1016/j.det.2012.08.005. [DOI] [PubMed] [Google Scholar]Banka N., Bunagan M.J., Shapiro J. Pattern hair loss in men: Diagnosis and medical treatment. Dermatol Clin. 2013;31:129140. doi: 10.1016/j.det.2012.08.003. [DOI] [PubMed] [Google Scholar]Birch M.P., Messenger J.F., Messenger A.G. Hair density, hair diameter and the prevalence of female pattern hair loss. Br J Dermatol. 2001;144:297304. doi: 10.1046/j.1365-2133.2001.04018.x. [DOI] [PubMed] [Google Scholar]Birch M.P., Lalla S.C., Messenger A.G. Female pattern hair loss. Clin Exp Dermatol. 2002;27:383388. doi: 10.1046/j.1365-2230.2002.01085.x. [DOI] [PubMed] [Google Scholar]Blume-Peytavi U., Orfanos C.E. Microscopy of the hair the trichogram. In: Serup J., Jemec G.B.E., Grove G.L., editors. Handbook of non-invasive methods and the skin. 2nd ed. CRC Press; Boca Raton, FL: 2006. [Google Scholar]Blume-Peytavi U., Hillmann K., Dietz E., Canfield D., Garcia Bartels N. A randomized, single-blind trial of 5% minoxidil foam once daily versus 2% minoxidil solution twice daily in the treatment of androgenetic alopecia in women. J Am Acad Dermatol. 2011;65:11261134. doi: 10.1016/j.jaad.2010.09.724. [DOI] [PubMed] [Google Scholar]Blume-Peytavi U., Lonnfors S., Hillmann K., Garcia Bartels N. A randomized double-blind placebo-controlled pilot study to assess the efficacy of a 24-week topical treatment by latanoprost 0.1% on hair growth and pigmentation in healthy volunteers with androgenetic alopecia. J Am Acad Dermatol. 2012;66:794800. doi: 10.1016/j.jaad.2011.05.026. [DOI] [PubMed] [Google Scholar]Blumeyer A., Tosti A., Messenger A., Reygagne P., Del Marmol V., Spuls P.I. Evidence-based (S3) guideline for the treatment of androgenetic alopecia in women and in men. J Dtsch Dermatol Ges. 2011;(Suppl. 6):S157. doi: 10.1111/j.1610-0379.2011.07802.x. [DOI] [PubMed] [Google Scholar]Bregy A., Trueb R.M. No association between serum ferritin levels >10 microg/l and hair loss activity in women. Dermatology. 2008;217:16. doi: 10.1159/000118505. [DOI] [PubMed] [Google Scholar]Camacho F., Tosti A. Tratamiento mdico de las alopecias femeninas. Monogr Dermatol. 2005;18:92117. [Google Scholar]Carey A.H., Chan K.L., Short F., White D., Williamson R., Franks S. Evidence for a single gene effect causing polycystic ovaries and male pattern baldness. Clin Endocrinol. 1993;38:653658. doi: 10.1111/j.1365-2265.1993.tb02150.x. [DOI] [PubMed] [Google Scholar]Carmina E., Lobo R.A. Treatment of hyperandrogenic alopecia in women. Fertil Steril. 2003;79:9195. doi: 10.1016/s0015-0282(02)04551-x. [DOI] [PubMed] [Google Scholar]Carter M.J., Fylling C.P., Parnell L.K. Use of platelet rich plasma gel on wound healing: A systematic review and meta-analysis. Eplasty. 2011;11:38. [PMC free article] [PubMed] [Google Scholar]Cervantes J., Perper M., Wong L.L., Eber A.E., Villasante Fricke A.C., Wikramanayake T.C. Effectiveness of platelet-rich plasma for androgenetic alopecia: A review of the literature. Skin Appendage Disord. 2018;4:111. doi: 10.1159/000477671. [DOI] [PMC free article] [PubMed] [Google Scholar]Dawber R.P., Sonnex T., Ralfs I. Oral antiandrogen treatment of common baldness in women. Br J Dermatol. 1982;107:2021. [Google Scholar]Deloche C., Bastien P., Chadoutaud S., Galan P., Bertrais S., Hercberg S. Low iron stores: A risk factor for excessive hair loss in non menopausal women. Eur J Dermatol. 2007;17:507512. doi: 10.1684/ejd.2007.0265. [DOI] [PubMed] [Google Scholar]Demay M.B. The hair cycle and vitamin D receptor. Arch Biochem Biophys. 2012;523:1921. doi: 10.1016/j.abb.2011.10.002. [DOI] [PubMed] [Google Scholar]Dhurat R., Sukesh M. Principles and methods of preparation of platelet-rich plasma: A review and author's perspective. J Cutan Aesthet Surg. 2014;7:189197. doi: 10.4103/0974-2077.150734. [DOI] [PMC free article] [PubMed] [Google Scholar]El Sayed M.H., Abdallah M.A., Aly D.G., Khater N.H. Association of metabolic syndrome with female pattern hair loss in women: A case-control study. Int J Dermatol. 2016;55:11311137. doi: 10.1111/ijd.13303. [DOI] [PubMed] [Google Scholar]Emer J.J., Stevenson M.L., Markowitz O. Novel treatment of female-pattern androgenetic alopecia with injected bimatoprost 0.03% solution. J Drugs Dermatol. 2011;10:795798. [PubMed] [Google Scholar]Fabi S., Sundaram H. The potential of topical and injectable growth factors and cytokines for skin rejuvenation. Facial Plast Surg. 2014;30:157171. doi: 10.1055/s-0034-1372423. [DOI] [PubMed] [Google Scholar]Fertig R.M., Gamret A.C., Cervantes J., Tosti A. Microneedling for the treatment of hair loss? J Eur Acad Dermatol Venereol. 2018;32:564569. doi: 10.1111/jdv.14722. [DOI] [PubMed] [Google Scholar]Fischer T.W., Scholz G., Knoll B. Melatonin reduces UV-induced reactive oxygen species in a dose-dependent manner in IL-3-stimulated leukocytes. J Pineal Res. 2001;31:3945. doi: 10.1034/j.1600-079x.2001.310106.x. [DOI] [PubMed] [Google Scholar]Fischer T.W., Slominski A., Tobin D.J., Paus R. Melatonin and the hair follicle. J Pineal Res. 2008;44:115. doi: 10.1111/j.1600-079X.2007.00512.x. [DOI] [PubMed] [Google Scholar]Fischer T.W., Trueb R.M., Hanggi G., Innocenti M., Elsner P. Topical melatonin for treatment of androgenetic alopecia. Int J Trichology. 2012;4:236245. doi: 10.4103/0974-7753.111199. [DOI] [PMC free article] [PubMed] [Google Scholar]Garza L.A., Liu Y., Yang Z., Alagesan B., Lawson J.A., Norberg S.M. Prostaglandin D2 inhibits hair growth and is elevated in bald scalp of men with androgenetic alopecia. Sci Transl Med. 2012;4 doi: 10.1126/scitranslmed.3003122. [DOI] [PMC free article] [PubMed] [Google Scholar]Goren A., Shapiro J., Roberts J., McCoy J., Desai N., Zarrab Z. Clinical utility and validity of minoxidil response testing in androgenetic alopecia. Dermatol Ther. 2015;28:1316. doi: 10.1111/dth.12164. [DOI] [PubMed] [Google Scholar]Gupta A.K., Foley K.A. 5% minoxidil: Treatment for female pattern hair loss. Skin Therapy Lett. 2014;19:57. [PubMed] [Google Scholar]Hajheydari Z., Akbari J., Saeedi M., Shokoohi L. Comparing the therapeutic effects of finasteride gel and tablet in treatment of the androgenetic alopecia. Indian J Dermatol Venereol Leprol. 2009;75:4751. doi: 10.4103/0378-6323.45220. [DOI] [PubMed] [Google Scholar]Heilmann-Heimbach S., Herold C., Hochfeld L.M., Hillmer A.M., Nyholt D.R., Hecker J. Meta-analysis identifies novel risk loci and yields systematic insights into the biology of male-pattern baldness. Nat Commun. 2017;8 doi: 10.1038/ncomms14694. [DOI] [PMC free article] [PubMed] [Google Scholar]Herskovitz I., Tosti A. Female pattern hair loss. Int J Endocrinol Metab. 2013;11(4) doi: 10.5812/ijem.9860. [DOI] [PMC free article] [PubMed] [Google Scholar]Hillmer A.M., Flaquer A., Hanneken S., Eigelshoven S., Kortm A.K., Brockschmidt F.F. Genome-wide scan and fine-mapping linkage study of androgenetic alopecia reveals a locus on chromosome 3q26. Am J Hum Genet. 2008;82:737743. doi: 10.1016/j.ajhg.2007.11.014. [DOI] [PMC free article] [PubMed] [Google Scholar]Iorizzo M., Vincenzi C., Voudouris S., Piraccini B.M., Tosti A. Finasteride treatment of female pattern hair loss. Arch Dermatol. 2006;142:298302. doi: 10.1001/archderm.142.3.298. [DOI] [PubMed] [Google Scholar]Jimenez J.J., Wikramanayake T.C., Bergfeld W., Hordinsky M., Hickman J.G., Hamblin M.R. Efficacy and safety of a low-level laser device in the treatment of male and female pattern hair loss: A multicenter, randomized, sham device-controlled, double-blind Study. Am J Clin Dermatol. 2014;15:115127. doi: 10.1007/s40257-013-0060-6. [DOI] [PMC free article] [PubMed] [Google Scholar]Jung J.Y., Yeon J.H., Choi J.W., Kwon S.H., Kim B.J., Youn S.W. Effect of dutasteride 0.5 mg/d in men with androgenetic alopecia recalcitrant to finasteride. Int J Dermatol. 2014;53:13511357. doi: 10.1111/ijd.12060. [DOI] [PubMed] [Google Scholar]Keaney T. Emerging therapies for androgenetic alopecia. J Drugs Dermatol. 2015;14:10361040. [PubMed] [Google Scholar]Lacarrubba F., Micali G., Tosti A. Scalp dermoscopy or trichoscopy. Curr Probl Dermatol. 2015;47:2132. doi: 10.1159/000369402. [DOI] [PubMed] [Google Scholar]Leavitt M., Charles G., Heyman E., Michaels D. HairMax LaserComb laser phototherapy device in the treatment of male androgenetic alopecia: A randomized, double-blind, sham device-controlled, multicentre trial. Clin Drug Investig. 2009;29:283292. doi: 10.2165/00044011-200929050-00001. [DOI] [PubMed] [Google Scholar]Lee S.H., Zheng Z., Kang J.S., Kim D.Y., Oh S.H., Cho S.B. Therapeutic efficacy of autologous platelet-rich plasma and polydeoxyribonucleotide on female pattern hair loss. Wound Repair Regen. 2015;23:3036. doi: 10.1111/wrr.12250. [DOI] [PubMed] [Google Scholar]Leo M.S., Kumar A.S., Kirit R., Konathan R., Sivamani R.K. Systematic review of the use of platelet-rich plasma in aesthetic dermatology. J Cosmet Dermatol. 2015;14(4):315323. doi: 10.1111/jocd.12167. [DOI] [PubMed] [Google Scholar]Ludwig E. Classification of the types of androgenetic alopecia (common baldness) occurring in the female sex. Br J Dermatol. 1977;97:247254. doi: 10.1111/j.1365-2133.1977.tb15179.x. [DOI] [PubMed] [Google Scholar]McCoy J., Kovacevic M., Situm M., Stanimirovic A., Bolanca Z., Goren A. Doppler laser imaging predicts response to topical minoxidil in the treatment of female pattern hair loss. J Biol Regul Homeost Agents. 2016;30:131134. [PubMed] [Google Scholar]McDonald K.A., Shelley A.J., Colantonio S., Beecker J. Hair pull test: Evidence-based update and revision of guidelines. J Am Acad Dermatol. 2017;76:472477. doi: 10.1016/j.jaad.2016.10.002. [DOI] [PubMed] [Google Scholar]Miao Y., Sun Y.B., Sun X.J., Du B.J., Jiang J.D., Hu Z.Q. Promotional effect of platelet-rich plasma on hair follicle reconstitution in vivo. Dermatol Surg. 2013;39:18681876. doi: 10.1111/dsu.12292. [DOI] [PubMed] [Google Scholar]Nieves A., Garza L.A. Does prostaglandin D2 hold the cure to male pattern baldness? Exp Dermatol. 2014;23:224227. doi: 10.1111/exd.12348. [DOI] [PMC free article] [PubMed] [Google Scholar]Olsen E.A. The midline part: An important physical clue to the clinical diagnosis of androgenetic alopecia in women. J Am Acad Dermatol. 1999;40:106109. doi: 10.1016/s0190-9622(99)70539-6. [DOI] [PubMed] [Google Scholar]Olsen E.A. Female pattern hair loss. J Am Acad Dermatol. 2001;45:S70S80. doi: 10.1067/mjd.2001.117426. [DOI] [PubMed] [Google Scholar]Olsen E.A. Female pattern hair loss. J Am Acad Dermatol. 2002;45:S70S80. doi: 10.1067/mjd.2001.117426. [DOI] [PubMed] [Google Scholar]Olsen E.A., Hordinsky M., Whiting D., Stough D., Hobbs S., Ellis M.L., Wilson T., Rittmaster R.S., Dutasteride Alopecia Research Team The importance of dual 5alpha-reductase inhibition in the treatment of male pattern hair loss: results of a randomized placebo-controlled study of dutasteride versus finsteride. J Am Acad Dermatol. 2006;55:10141023. doi: 10.1016/j.jaad.2006.05.007. [DOI] [PubMed] [Google Scholar]Orme S., Cullen D.R., Messenger A.G. Diffuse female hair loss: Are androgens necessary? Br J Dermatol. 1999;141:521523. doi: 10.1046/j.1365-2133.1999.03049.x. [DOI] [PubMed] [Google Scholar]Pereira J.O., Pereira Filho J.O., Cabrera Pereira J.O. Mega-sessions for robotic hair restoration. J Drugs Dermatol. 2016;15:14071412. [PubMed] [Google Scholar]Pickrell J.K., Berisa T., Liu J.Z., Segureal L., Tung J.Y., Hinds D.A. Detection and interpretation of shared genetic influences on 42 human traits. Nat Genet. 2016;48:709717. doi: 10.1038/ng.3570. [DOI] [PMC free article] [PubMed] [Google Scholar]Pirard G.E., Pirard-Franchimont C., NikkelsTassoudji N., Nikkels A.F., Saint Leger D. Improvement in the inflammatory aspect of androgenetic alopecia: A pilot study with an antimicrobial lotion. J Dermatol Treat. 1996;7:153157. [Google Scholar]Pirard-Franchimont C., De Doncker P., Cauwenbergh G., Pirard G.E. Ketoconazole shampoo: Effect of long-term use in androgenic alopecia. Dermatology. 1998;196:474477. doi: 10.1159/000017954. [DOI] [PubMed] [Google Scholar]Price V.H., Roberts J.L., Hordinsky M., Olsen E.A., Savin R., Bergfeld W. Lack of efficacy of finasteride in post-menopausal women with androgenetic alopecia. J Am Acad Dermatol. 2000;43:768776. doi: 10.1067/mjd.2000.107953. [DOI] [PubMed] [Google Scholar]Ramos P.M., Miot H.A. Female pattern hair loss: A clinical and pathophysiological review. An Bras Dermatol. 2015;90(4):529543. doi: 10.1590/abd1806-4841.20153370. [DOI] [PMC free article] [PubMed] [Google Scholar]Randall V.A. Androgens and hair growth. Dermatol Ther. 2008;21:314328. doi: 10.1111/j.1529-8019.2008.00214.x. [DOI] [PubMed] [Google Scholar]Rangwala S., Rashid R.M. Alopecia: A review of laser and light therapies. Dermatol Online J. 2012;18:3. [PubMed] [Google Scholar]Rasheed H., Mahgoub D., Hegazy R., El-Komy M., Abdel Hay R., Hamid M.A. Serum ferritin and vitamin d in female hair loss: Do they play a role? Skin Pharmacol Physiol. 2013;26:101107. doi: 10.1159/000346698. [DOI] [PubMed] [Google Scholar]Rebora A., Guarrera M., Baldari M., Vecchio F. Distinguishing androgenetic alopecia from chronic telone effluvium when associated in the same patient. Arch Dermatol. 2005;143:12431245. doi: 10.1001/archderm.141.10.1243. [DOI] [PubMed] [Google Scholar]Redler S., Messenger A.G., Betz R.C. Genetics and other factors in the aetiology of female pattern hair loss. Exp Dermatol. 2017;26:510517. doi: 10.1111/exd.13373. [DOI] [PubMed] [Google Scholar]Rose P.T. The latest innovations in hair transplantation. Facial Plast Surg. 2011;27:366377. doi: 10.1055/s-0031-1283055. [DOI] [PubMed] [Google Scholar]Rushton D.H., Ramsay I.D. The importance of adequate serum ferritin levels during oral cyproterone acetate and ethinyl oestradiol treatment of diffuse androgen-dependent alopecia in women. Clin Endocrinol. 1992;36:421427. doi: 10.1111/j.1365-2265.1992.tb01470.x. [DOI] [PubMed] [Google Scholar]Savin R.C. The Upjohn Company; Kalamazoo, MI: 1994. Evaluating androgenetic alopecia in male and female patients. [Google Scholar]Shah K.B., Shah A.N., Solanki R.B., Raval R.C. A comparative study of microneedling with platelet-rich plasma plus topical minoxidil (5%) and topical minoxidil (5%) alone in androgenetic alopecia. Int J Trichology. 2017;9:1418. doi: 10.4103/ijt.ijt_75_16. [DOI] [PMC free article] [PubMed] [Google Scholar]Shum K.W., Cullen D.R., Messenger A.G. Hair loss in women with hyperandrogenism: Four cases responding to finasteride. J Am Acad Dermatol. 2002;47:733739. doi: 10.1067/mjd.2002.124608. [DOI] [PubMed] [Google Scholar]Sinclair R.D., Dawber R.P. Androgenetic alopecia in men and women. Clin Dermatol. 2001;19:167178. doi: 10.1016/s0738-081x(00)00128-0. [DOI] [PubMed] [Google Scholar]Sinclair R., Wewerinke M., Jolley D. Treatment of female pattern hair loss with oral antiandrogens. Br J Dermatol. 2005;152:466473. doi: 10.1111/j.1365-2133.2005.06218.x. [DOI] [PubMed] [Google Scholar]Sinclair R., Jolley D., Mallari R., Magee J. The reliability of horizontally sectioned scalp biopsies in the diagnosis of chronic diffuse telogen hair loss in women. J Am Acad Dermatol. 2004;51:189199. doi: 10.1016/s0190-9622(03)00045-8. [DOI] [PubMed] [Google Scholar]Sinclair R.D. Female pattern hair loss: A pilot study investigating combination therapy with low-dose oral minoxidil and spironolactone. Int J Dermatol. 2018;57:104109. doi: 10.1111/ijd.13838. [DOI] [PubMed] [Google Scholar]Singal A., Sonthalia S., Verma P. Female pattern hair loss. Indian J Dermatol Venereol Leprol. 2013;79:626640. doi: 10.4103/0378-6323.116732. [DOI] [PubMed] [Google Scholar]Singh M., Jadhav H.R. Melatonin: Functions and ligands. Drug Discov Today. 2014;19:14101418. doi: 10.1016/j.drudis.2014.04.014. [DOI] [PubMed] [Google Scholar]Sonino N., Scaroni C., Biason A., Boscaro M., Mantero F. Low-dose ketoconazole treatment in hirsute women. J Endocrinol Investig. 1990;13:3540. doi: 10.1007/BF03348578. [DOI] [PubMed] [Google Scholar]Stefanato C.M. Histopathology of alopecia: A clinicopathological approach to diagnosis. Histopathology. 2010;56:2438. doi: 10.1111/j.1365-2559.2009.03439.x. [DOI] [PubMed] [Google Scholar]Takikawa M., Nakamura S., Nakamura S., Ishirara M., Kishimoto S., Sasaki K. Enhanced effect of platelet-rich plasma containing a new carrier on hair growth. Dermatol Surg. 2011;37:17211729. doi: 10.1111/j.1524-4725.2011.02123.x. [DOI] [PubMed] [Google Scholar]Thai K.E., Sinclair R.D. Finasteride for female androgenetic alopecia. Br J Dermatol. 2002;147:812813. doi: 10.1046/j.1365-2133.2002.49084.x. [DOI] [PubMed] [Google Scholar]Tosti A., Piraccini B.M. Androgenetic alopecia. In: Tosti A., Piraccini B.M., editors. Diagnosis and treatment of hair disorders: An evidence based atlas. Taylor and Francis; London, UK: 2006. [Google Scholar]Tsuboi R., Arano O., Nishikawa T., Yamada H., Katsuoka K. Randomized clinical trial comparing 5% and 1% topical minoxidil for the treatment of androgenetic alopecia in Japanese men. J Dermatol. 2009;36:437446. doi: 10.1111/j.1346-8138.2009.00673.x. [DOI] [PubMed] [Google Scholar]Valente Duarte de Sousa I.C., Tosti A. New investigational drugs for androgenetic alopecia. Expert Opin Investig Drugs. 2013;22:573589. doi: 10.1517/13543784.2013.784743. [DOI] [PubMed] [Google Scholar]Varothai S., Bergfeld W.F. Androgenetic alopecia: An evidence-based treatment update. Am J Clin Dermatol. 2014;15:217230. doi: 10.1007/s40257-014-0077-5. [DOI] [PubMed] [Google Scholar]Vexiau P., Chaspoux C., Boudou P., Fiet J., Jouanique C., Hardy N. Effects of minoxidil 2% vs. cyproterone acetate treatment on female androgenetic alopecia: A controlled, 12-month randomized trial. Br J Dermatol. 2002;146:992999. doi: 10.1046/j.1365-2133.2002.04798.x. [DOI] [PubMed] [Google Scholar]Vujovic A., Del Marmol V. The female pattern hair loss: Review of etiopathogenesis and diagnosis. Biomed Res Int. 2014;2014 doi: 10.1155/2014/767628. [DOI] [PMC free article] [PubMed] [Google Scholar]Yip Y., Sinclair R.D. Antiandrogen therapy for androgenetic alopecia. Expert Rev Dermatol. 2006;1:261269. [Google Scholar]van Zuuren E.J., Fedorowicz Z., Carter B. Evidence-based treatments for female pattern hair loss: A summary of a Cochrane systematic review. Br J Dermatol. 2012;167:9951010. doi: 10.1111/j.1365-2133.2012.11166.x. [DOI] [PubMed] [Google Scholar]van Zuuren E.J., Fedorowicz Z., Carter B., Andriolo R.B., Schoones J. Interventions for female pattern hair loss. Cochrane Database Syst Rev. 2012;5 doi: 10.1002/14651858.CD007628.pub3. [DOI] [PubMed] [Google Scholar]Articles from International Journal of Women's Dermatology are provided here courtesy of Wolters Kluwer Health

Female pattern baldness treatment in ayurveda. Female pattern baldness treatment natural. Female pattern baldness treatment reddit. Female pattern baldness homeopathy treatment. New treatment for female pattern baldness. Female pattern baldness treatment near me. Frontal female pattern baldness treatment. Female pattern baldness treatment at home. Prp hair treatment for female pattern baldness. Female pattern baldness treatment nhs. Which medication is used for the treatment of both male and female pattern baldness. Female pattern baldness in 20s treatment. Prp treatment for female pattern baldness. Ayurvedic treatment for female pattern baldness. Female pattern baldness treatment uk.