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Antibiotic Resistance at TTH Clinical and Biomedical Evidence; Collection and Analysis of Sputum Culture Sensitivity of Patients of Chest Clinic

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ABSTRACT

Introduction: *Symbiotic resistance means two or more microbial species working together as a group to cause resistance.*

Previous research finding noted that Moraxella catarrhalis was found in more than 30% of tested TB patients of TTH

In this research, the microbes are mycobacteria TB assisted by nosocomial infection

Methodology: *Retrospective analysis of sputum microscopy results of patients of pulmonary tuberculosis was done from 2014 to 2018 at the bacteriology department of the Tamale Teaching Hospital.*

Results: *Citrobacter spp was found to be the most prevalent 53%, followed by Commensals Only and Citrobacter diversus.*

The clinical and bacterial resistance persisted until the right (attacking doses) of antibiotics were used to prevent extended spectrum of β lactamase effect.

This was a confirmation of the fact that holistic treatment (use of combined treatment) is the most effective clinical practice against tuberculosis.

Conclusion: *Nosocomial coinfection resulted to symbiotic resistance of mycobacteria TB in the form of extended spectrum of β lactamase.*

Recommendations: *Early intervention and treatment before complication*

Wholistic treatment to all patients

Appropriate antibiotics

Appropriate dosages and appropriate timing of treatment

BACKGROUND

The rapid emergence of resistant bacteria is occurring worldwide, endangering the efficacy of antibiotics, which have transformed medicine and saved millions of lives. Many decades after the first patients were treated with antibiotics; bacterial infections have again become a threat. The antibiotic resistance crisis has been attributed to the overuse and misuse of these medications, as well as a lack of new drug development by the pharmaceutical industry due to reduced economic incentives and challenging regulatory requirements. The Centres for Disease Control and Prevention (CDC) has classified a number of bacteria as presenting urgent, serious, and concerning threats, many of which are already responsible for placing a

Substantial clinical and financial burden on the Ghanaian health care system, patients, and their families. Coordinated efforts to implement new policies, renew research efforts, and pursue steps to manage the crisis are greatly needed. The term “antibiotics” was first coined by the American microbiologist Selman Waksman and his colleagues to describe chemical substances produced by microorganisms and having antagonistic effects on the growth of other microorganisms. It excluded synthetic antimicrobials (sulphur drugs) and biological products of non-microbial origin having

antagonistic effects on bacteria. Though antibiotics were introduced into the clinical practice only in the middle of the last century, the use of microorganisms for the management of microbial infections in ancient Egypt, Greece, China, and some other places of the world is well-documented. The modern era of antibiotics started with the serendipitous discovery of penicillin from the culture filtrate of a fungus, Penicillium notatum by Alexander Fleming in 1928 (Fleming, 1929). In the present scenario, antibiotics available in the market are either produced by microbial fermentation or are derived via semi-synthetic route using the existing antibiotic backbone structure. They are classified into different chemically defined groups. Antibiotics target bacterial physiology and biochemistry, causing microbial cell death or the cessation of growth. A significant number of these antibiotics affect cell walls or membranes (e.g., β-lactam and glycopeptides), while several others exert their antibacterial activity by targeting protein synthetic machinery via interaction with ribosomal subunits and these include antibiotics such as macrolides, chloramphenicol, tetracycline, linezolid, and aminoglycosides. Other "mechanistic" groups include molecules which interfere with the nucleic acid synthesis [e.g., fluoroquinolones (FQ) and rifampin], while some others exert their effects by interfering with the metabolic pathways (e.g., sulphonamides and folic acid analogue) or by disruption of the bacterial membrane structure (e.g., polymyxins, daptomycin, and others). A surge of discovery of several such antibacterial and antifungal antibiotics accompanied with a new generation of semisynthetic drugs initially led to euphoria that any infectious disease could be successfully controlled using antibiotics. However, emergence and propagation of bacterial strains, resistant to almost all the therapeutically useful antibiotics during the past few decades revealed the limitation of the wonder drugs. Though imprudent and excessive use of antibiotics is highlighted as a major causative factor behind the setback, it is evident by this time that antibiotic resistance does not call for exposure of the organisms to antibiotics. It is also found that genes involved in the biosynthesis of antibiotics and antibiotic resistance evolved thousands of years before antibiotics were introduced into the clinical practice. Hence both antibiotic and its resistance determinants have some other role in bacterial physiology.

ANTIBIOTIC RESISTANCE AT TAMALE TEACHING HOSPITAL (TTH): Clinical and Biomedical Evidence; Collection And Analysis Of Sputum Culture and Sensitivity Of Patients Of Chest Clinic.

INTRODUCTION

Antibiotic resistance is the ability of bacteria to resist the harmful effects of an antibiotic to which it was susceptible initially. In other words, the antibiotic has now lost its ability to destroy or stop the growth of the bacteria. The bacteria is said to be resistant to the antibiotic and continue to grow in the presence of adequate amounts of the antibiotic. Resistance can be intrinsic (exist in the microorganism before exposure to the antibiotic) or acquired as a result of subsequent exposure to an antibiotic.

About Antibiotic Resistance

Bacteria resistance to antibiotics is a natural phenomenon. When antibiotics are used mostly incorrectly, some susceptible bacteria are destroyed while other strains that can withstand the particular antibiotic survive and multiply. This results in what is referred to as selective pressure for the survival of the resistant strains. Antibiotics can also destroy friendly bacteria which would otherwise compete with the resistant strain for the same resources to survive, making the resistant strain multiply.

Resistance by bacteria can be acquired from other bacteria through resistant genes; by mutation through changes in genetic material; changes in the way the bacteria handles the antibiotic (eg. Reduced penetration or degradation of antibiotics); or, may be an intrinsic natural phenomenon. Almost every type of bacteria responsible for the common infection in man have become stronger and less responsive to antibiotic treatment as a result of resistance.

These antibiotic-resistant bacteria can quickly spread to families and members of the community, thereby threatening the emergence of a new strain of infectious disease that may be more difficult and more expensive to treat. Resistant bacteria can also spread from one host to the other directly or indirectly through contact, food, the environment, etc.

The spread of resistance may be linked to the use of antibiotics; the more we use antibiotics for conditions that do not require them, the more there is selective pressure and the more resistance strains we get.

Can bacteria lose their antibiotic resistance?

If an antibiotic is reserved for a period of time, it can possibly regain its action on the resistant bacteria traits. This however is a slow process.

If the selective pressure that is applied by the presence of an antibiotic is removed, the bacterial population can potentially revert a population that responds to antibiotics.

Research Questions

1. What is the prevalence of antibiotic resistance at the Tamale Teaching Hospital?
2. What is the mode/trend of antibiotic resistance at TTH?
3. Can bacteria lose their antibiotic resistance?
4. What are the factors that promote antibiotic resistance at TTH?
5. The impact ofantibiotic resistance at TTH?

Objectives

1. The main objective of the research is to determine the prevalence of antibiotic resistance at TTH.
2. To determine the factors that promotes antibiotic resistance at TTH.
3. To determine the mode/trend of antibiotic resistance at TTH.
4. To determine the consequences of antibiotic resistance at TTH.

LITERATURE REVIEW

From our previous research entitled 'Antibiotic Resistance Pattern of Moraxella Catarrhalis in Patients with Respiratory Tract Infection at Tamale Teaching Hospital', it was documented that, the large majority of antibiotics currently used for treating infections and the antibiotic resistance genes acquired by human pathogens each have an environmental origin. Recent work indicates that the function of these elements in their environmental reservoirs may be very distinct from the "weapon-shield" role they play in clinical settings. Changes in natural ecosystems, including the release of large amounts of antimicrobials, might alter the population dynamics of microorganisms, including selection of resistance, with consequences for human health that are difficult to predict (WHO 2003).

From the same research it was found that both antibiotic biosynthetic genes and resistance-conferring genes have been known to evolve many years ago, long before clinical use of antibiotics. Hence it appears that antibiotics and antibiotics resistance determinants have some other roles in nature, which often elude our attention because of overemphasis on the therapeutic importance of antibiotics and the crisis imposed by the antibiotic resistance in pathogens. In the natural milieu, antibiotics are often found to be present in sub-inhibitory concentrations acting as signalling molecules supporting the process of quorum sensing and biofilm formation. They also play an important role in the production of virulence factors and influence host–parasite interactions (example; phagocytosis, adherence to the target cell, and so on). The evolutionary and ecological aspects of antibiotics and antibiotic resistance in the naturally occurring microbial community are little understood (Yahaya Abdallah, 2018. ISSN No. (Online) +2332456-0596).

From the workshop of Civil Society Organization on Community education in 2010, it was stated that Resistance by bacteria can be acquired from other bacteria through resistant genes; by mutation through changes in genetic material; changes in the way the bacteria handles the antibiotic (example; Reduced penetration or degradation of antibiotic); or, may be an intrinsic natural phenomenon. Almost every type of bacteria responsible for the common infection in man has become stronger and less responsive to antibiotic treatment as a result of resistance.

According to WHO 2010, data in Ghana shows that, resistance to antibiotics is on the increase, especially in the commonly used antibiotics, it has also been shown that, some bacteria have developed resistance to the current and expensive antibiotics being used in the health system. Furthermore, the development of antibiotics with novel action has been stalled over the years. This phenomenon has become a global public health concern as antibiotics resistance threatens the modest gains made in infectious disease therapy.

Findings from a research by Noguchi Memorial Institute in 2019;Most of the M. bovis isolates (13) were susceptible to all the drugs tested except two of isolates resistant to INH and one isolate resistant to RIF (table1). The two INH resistant isolates both had S315Tmutation in *katG* while the RIF resistant isolate had Q432p and I1491S mutations in rpoB. They identified

41 M. restricted amino acid mutations among 32 core-genes of the clinical M. bovis from Ghana when compared to 257 *maf*[27] and 20 global MTBC genomes [41]. (Otchirel 2018)

The modern era of antibiotics started with the serendipitous discovery of penicillin from the culture filtrate of a fungus, Penicillium notatum by Alexander Fleming in 1928 (Fleming, 1929).

A significant number of these antibiotics affect cell walls or membranes (e.g., β-lactam and glycopeptides), while several others exert their antibacterial activity by targeting protein synthetic machinery via interaction with ribosomal subunits and these include antibiotics such as macrolides, chloramphenicol, tetracycline, linezolid, and aminoglycosides. Other "mechanistic" groups include molecules which interfere with the nucleic acid synthesis [e.g., fluoroquinolones (FQ) and rifampin], while some others exert their effects by interfering with the metabolic pathways (e.g., sulphonamides and folic acid analogue) or by disruption of the bacterial membrane structure (e.g., polymyxins, daptomycin, and others).

Drug Resistance Profile of M. Bovis Isolates

Most of the M. bovis isolates(13) were susceptible to all the drugs tested except two of islolates resistant to INH and one isolate resistant to RIF (table1). The two INH resistant isolates both had S315Tmutation in *katG* while the RIF resistant isolate had Q432p and I1491Smutations in rpoB.

In Silico M. Bovis-Specific Amino Acid Mutations

We identified 41 M. restricted amino acid mutations among 32 core-genes of the clinical M. bovis from Ghana when compared to 257 *maf*[27] and 20 global MTBC genomes [41].

Among the 41 mutations identified uniquely among 32 core-genes M. bovis, 17 were among 15 essential associated with important physiological processes such as lipid metabolism wall and cell processes, intermediate metabolism, and cellular respiration, virulence detoxification and virulence as well as regulatory proteins (table2). These include *mceA, phoT and eccA1*previously shown to be for the growth of *mtbss L4* strain H37Rv primary murine macrophages [35]. In additions in other genes such as pks6, pks4 and glgP have been shown to be associated with no production of phthiocerol dimycocerosates (PDIM) among mutant strains [36], attenuation in the central nervous system of BALB/c mice [39], no production of mycolic acid derivatives (mycolipanic, mycolipenicanndmycolipodienoic acids) among mutant strains [38] and in vitro sow growth [34]. (Okyere Babra et al, 2017)

METHODOLOGY

Cross-sectional approach

Primary data from clinical retrospective review of secondary data collected as records of patients from specialist clinic of Tamale Teaching Hospital.

Retrospective review of records of patients from biomedical data in the laboratory of TTH.

RESULTS: Retrospective Analysis of Culture And Sensitivity Results Of Sputum Microscopy of Patients at Chest Clinic of TTH.

Table1. Distribution of Pathogens and their percentages from the bacteriology department of TTH.

ISOLATE 1	SENSITIVE	RESISTANT	INTERMEDIATE	TOTAL COUNTS	PERCENTAGE
Pseudomonas aeruginosa	56	90	0	146	5.16631281
Moraxella Catarrhalis	452	661	8	1121	39.66737438
Proteus vulgaris	9	15	0	24	0.8492569
Morganella moganii	14	25	0	39	1.380042463
Enterobacter spp	152	164	3	319	11.28803963
Pseudomonas spp	38	48	0	86	3.043170559
Proteus spp	6	9	0	15	0.530785563
Citrobacter spp	74	63	0	137	4.847841472
Streptococcus pneumoniae	12	12	0	24	0.8492569
Klebsiella spp	242	239	1	482	17.05590941
Enterococcus spp	28	44	0	72	2.547770701

Citrobacter diversus	8	10	0	18	0.636942675
Escherichia coli	44	61	0	105	3.715498938
Proteus mirabilis	24	26	0	50	1.769285209
Klebsiella oxytoca	40	50	0	90	3.184713376
Serratia marcensces	3	6	0	9	0.212314225
Enterobacter species	2	4	0	6	0.212314225
Staphylococcus aureus	24	27	0	51	1.804670913
Klebsiella pneumonia	11	1	0	12	0.42462845
Coagulase negative Staphylococcus	2	10	0	12	0.42462845
Acinetobacter baumanii	3	5	0	8	0.283085633
TOTAL	**1247**	**1567**	**12**	**2826**	

Out of 2826 microbes tested, 1247 were resistant. This is biomedical confirmation of resistance of microbes to antibiotics at TTH.

Table2. *Summary of Sputum Smears (TB Microscopy) Examined by Facilities in the Northern Region Jan-Dec, 2014 by Abass, Public Health Lab TTH*

Facility	Total Examined	No. Positive	New Cases	No. Positive	Follow-up	No. Positive
Public Health Lab	78	5	74	4	4	1
Yendi Hospital	288	31	260	24	47	7
Walewale Hospital	176	12	155	10	21	2
Baptist Med. Centre	635	59	454	46	181	59
Salaga Hospital	131	39	169	33	62	6
Savelegu Hospital	267	14	254	12	13	2
Bole Hospital	35	2	33	1	2	1
WestGonja Hospital	77	8	64	5	13	3

Table 2 shows that a number patients after the treatment came for review with positive sputum results, in that case the anti kochs fails to destroy the microbes. As shown by Baptist Med. Centre 59 patients out of 635 had positive results and after treatment their review shows that same 59 being positive smear.

This results is a biomebical evidence of proven clinical resistance ; for af.ter 6 months DOTs (direct Observatory treatment), the sputum microscopy is expected to be Negetive, (WHO 2003. The positive results is evidence of Resistance

Table3. *Distribution of Pathogens and their percentages/sputum culture and sensitivity of 2,826 patients of chest clinic at TTH from 2017 to 2019*

Isolate 1	SENSITIVE	RESISTANT	INTERMEDIATE	TOTAL COUNTS	PERCENTAGE
Pseudomonas aeruginosa	56	90	0	146	5.16631281
Moraxella Catarrhalis	452	661	8	1121	39.66737438
Proteus vulgaris	9	15	0	24	0.8492569
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Proteus mirabilis	24	26	0	50	1.769285209
Klebsiella oxytoca	40	50	0	90	3.184713376
Serratia marcensces	3	6	0	9	0.212314225
Enterobacter species	2	4	0	6	0.212314225
Staphylococcus aureus	27	24	0	51	1.804670913
Klebsiella pneumonia	11	1	0	12	0.42462845
Isolate 1	Sensitive	Resistant	Intermediate	Total Counts	percentage
Coagulase negative Staphylococcus	2	10	0	12	0.42462845
Acinetobacter baumanii	3	5	0	8	0.283085633
TOTAL	**1247**	**1567**	**12**	**2826**	

Table4. *Clinical diagnosis of M. catarrhalis isolates*

Clinical Diagnosis	**Number**
Pneumonia	521
Acute Exacerbation of COPD	401
Chronic Bronchitis	199
TOTAL	1,121

Table5. *Antibiotic susceptibility pattern of Moraxella catarrhalis(1121)*

Antibiotics	**Sensitive (%)**	**Resistant (%)**
Ampicillin (10µg)	(31.27)	(68.72)
Chloramphenicol(15µg)	(83.33)	(15.66)
Gentamicin (10µg)	(84.33)	(15.66)
Cefotaxime (30µg)	(86.17)	(13.82)
Amoxicillin clavulanate (10/10µg)	(95.17)	(4.82)
Cotrimoxazole (1.25/23.75µg)	(81.41)	(18.58)
Ciprofloxacin (5µg)	(81.47)	(18.52)

Summary of Sputum Smears (TB Microscopy) Examined by Facilities in the Northern Region Jan-Dec,2014 By Abass, Public Health Lab

Facility	**Total Examined**	**No Positive**	**New Cases**	**No. Positive**	**Follow-up**	**No positive**
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Babtis Med. Centre	635	59	454	46	181	59
Salaga Hos	131	39	169	33	62	6
Savelugu Hos	267	14	254	12	13	2
Bole Hos	35	2	33	1	2	1
West Gonja Hos	77	8	64	5	13	3
Saboba Hos	**229**	**22**	**209**	**22**	**19**	**0**
Bimbila Hos	340	42	231	31	109	11
Gushigu Hos	143	27	136	25	7	2

Tamale West Hos	94	4	92	3	2	0
Tamale Central Hos	532	28	451	28	82	0
Kings Med. Centre	90	1	90	1	0	0
KaragaDist Hos	58	0	57	0	1	0
Zabzugu Hos	57	2	43	2	11	0
TOTAL	**3232**	**295**	**2739**	**245**	**572**	**95**

TABLE 3: CONFIRMATORY RESULTS OF MULTIPLE DRUG RESISTANT TB AT THE PUBLIC HEALTH LABORATORY AT TTH (GENE EXPERT ANALYSIS) 2013-2015.

Ghana Health Services Laboratory results on multiply drug resistance Tuberculosis in Northern Ghana. By Abass Karim.

Facility	Total No. of Samples	No. Positive
Tamale Teaching Hospital	11	2
Salaga Hospital	2	1
Tamale Central Hospital	7	5
Bole Hospital	1	0
Total	**21**	**8 (38.1%)**

DISCUSSION OF RESULTS

As found in table, Moraxella Catarrhalis causes pneumonia 521, Acute exacerbation 401 and chronic bronchitis 199 in patients.

This indicates that moraxale catarrhalis is nomore a commensal as stated in literature thirty years ago.

Also as found in table 5, the antibiotic susceptibility pattern of Moraxella catarrhalis was investigated and the findings was that the resistance pattern is high in almost all the first generation antibiotics as compared to the susceptibility. Ampicillin 68.72%, chloramphenicol 15.66%, Gentamicin 15.66%, Cefotaxime 13.82%, amoxicillin clavulantae 4.82%, cotrimoxazole 18.58%, and ciprofloxacin 18.52%. Amoxicillin clavulanate was the most susceptible with 95.17% and ampicillin was the most resistant antibiotic with 68.72% tested.

CONCLUSION

Moraxella catarrhalis was the most prevalent bacteria in the respiratory tract of patients of chest clinic at TTH. Its resistant pattern shows m catarrhalis is resistant against first generation antibiotics and some third generation cephalosspoirins. Attacking doses must be used to achieve sensitivity. Most antibiotics are intermediate in their action and have to be used appropriately to void creating resistance.

RECOMMENDATION

Appropriate choice of antibiotics, appropriate needed dosages, appropriate specific diagnosis and appropriate media of administration must be used to prevent resistance. Intensivepharmaco vigilance is empirical to avoid microbial resistance to antibiotics.

REFERENCES

[1] AAVMC (Association of American Veterinary Medical Colleges). Antimicrobial resistance: The AAVMC is taking strategic action to confront a critical threat to public health. 2017. [July 30, 2017]. http://www.aavmc.org/ Publications/ Antimicrobial-Resistance003A-Taking-StrategicAction-Against-a-Critical-Public-Health-Threat.aspx.

[2] Barlam TF, Cosgrove SE, Abbo LM, MacDougall C, Schuetz AN, Septimus EJ, Srinivasan A, Dellit TH, Falck-Ytter YT, Fishman NO, Hamilton CW, Jenkins TC, Lipsett PA, Malani PN, May LS, Moran GJ, Neuhauser MM, Newland JG, Ohl CA, Samore MH, Seo SK, Trivedi KK. Implementing an antibiotic stewardship program: Guidelines by the Infectious Diseases Society of America and the Society for Healthcare Epidemiology of America. Clinical Infectious Diseases. 2016; 62(10):e51–e77. [PMC free article] [PubMed]

[3] Barnett ML, Linder JA. Antibiotic prescribing to adults with sore throat in the United States, 1997-2010. JAMA Internal Medicine. 2014; 174(1):138–140. [PMC free article] [PubMed]

[4] Baur D, Gladstone BP, Burkert F, Carrara E, Foschi F, Dobele S, Tacconelli E. Effect of antibiotic stewardship on the incidence of infection and colonisation with antibiotic-resistant bacteria and Clostridium difficile infection: A systematic review and meta-analysis. Lancet Infectious Diseases. 2017;17(9):990–1001. [PubMed]

[5] Bilinski J, Grzesiowski P, Sorensen N, Madry K, Muszynski J, Robak K, Wroblewska M, Dzieciatkowski T, Dulny G, Dwilewicz-Trojaczek J, Wiktor-Jedrzejczak W, Basak GW. Fecal microbiota transplantation in

patients with blood disorders inhibits gut colonization with antibiotic-resistant bacteria: Results of a prospective, single-center study. Clinical Infectious Diseases. 2017; 65(3) :364–370. [PubMed]

[6] Caly DL, D'Inca R, Auclair E, Drider D. Alternatives to antibiotics to prevent necrotic enteritis in broiler chickens: A microbiologist's perspective. Frontiers in Microbiology. 2015; 6:1336. [PMC free article] [PubMed]

[7] CDC (U.S. Centers for Disease Control and Prevention). About antimicrobial resistance. 2013a. [November 6, 2017]. https://www.cdc.gov/drugresistance/about.html.

[8] CDC. Antibiotic resistance threats in the United States, 2013. Atlanta, GA: CDC; 2013b.

[9] CDC. Core elements of hospital antibiotic stewardship programs. Atlanta, GA: CDC; 2014.

[10] CDC. Measuring outpatient antibiotic prescribing. 2017. [July 30, 2017]. https://www.cdc.gov/getsmart/community/programs-measurement/measuring-antibiotic-prescribing.html.

[11] Davies J, Davies D. Origins and evolution of antibiotic resistance. Microbiology and Molecular Biology Review. 2010;74(3):417–433. [PMC free article] [PubMed]

[12] Dbaibo G, Claeys C, Izu A, Kosalaraksa P, Rivera L, Schuind A, Zaman K, Arroba Basanta ML, Aziz A, Cabanero MA, Chandrashekaran V, Corsaro B, Cousin L, Dinleyici EC, Faust SN, Mares Bermudez J, Martinon-Torres F, Miranda M, Montellano M, Peix Sambola MA, Prymula R, Puthanakit T, Ruzkova R, Sadowska-Krawczenko I, Salamanca de la Cueva I, Soni J, Szymanski H, Ulied A, Jain VK, Innis BL., Flu4VEC Study Group. Inactivated quadrivalent influenza vaccine (IIV4) reduces influenza-associated healthcare, antibiotic use, and parent-child absenteeism during a randomized controlled trial in healthy children aged 6-35 months. Madrid, Spain: May 23-27, 2017. 2017. Paper read at European Society for Paediatric Infectious Diseases.

[13] D'Costa VM, King CE, Kalan L, Morar M, Sung WW, Schwarz C, Froese D, Zazula G, Calmels F, Debruyne R, Golding GB, Poinar HN, Wright GD. Antibiotic resistance is ancient. Nature. 2011; 477(7365): 457–461. [PubMed]

[14] Duke-Margolis Center for Health Policy. Value-based strategies for encouraging new development of antimicrobial drugs. Washington, DC: Duke University; 2017.

[15] Fireman B, Black SB, Shinefield HR, Lee J, Lewis E, Ray P. Impact of the pneumococcal conjugate vaccine on otitis media. Pediatric Infectious Disease Journal. 2003;22(1):10–16. [PubMed]

[16] Fleming-Dutra KE, Hersh AL, Shapiro DJ, Bartoces M, Enns EA, File TM Jr., Finkelstein JA, Gerber JS, Hyun DY, Linder JA, Lynfield R, Margolis DJ, May LS, Merenstein D, Metlay JP, Newland JG, Piccirillo JF, Roberts RM, Sanchez GV, Suda KJ, Thomas A, Woo TM, Zetts RM, Hicks LA. Prevalence of inappropriate antibiotic prescriptions among U.S. ambulatory care visits, 2010-2011. JAMA. 2016;315(17):1864–1873. [PubMed] GARP-Kenya Working Group. Situation analysis and recommendations: Antibiotic use and resistance in Kenya. Washington, DC: The Center for Disease Dynamics, Economics & Policy; 2011.

[17] Gertz RE Jr., Li Z, Pimenta FC, Jackson D, Juni BA, Lynfield R, Jorgensen JH, Carvalho Mda G, Beall BW. Increased penicillin nonsusceptibility of nonvaccine-serotype invasive pneumococci other than serotypes 19A and 6A in post-7-valent conjugate vaccine era. Journal of Infectious Diseases. 2010;201(5):770–775. [PubMed]

[18] Government of Kenya. National policy on prevention and containment of antimicrobial resistance. Nairobi, Kenya: Ministry of Health and Ministry of Agriculture, Livestock & Fisheries; 2017.

[19] Hallsworth M, Chadborn T, Sallis A, Sanders M, Berry D, Greaves F, Clements L, Davies SC. Provision of social norm feedback to high prescribers of antibiotics in general practice: A pragmatic national randomised controlled trial. Lancet. 2016;387(10029):1743–1752. [PMC free article] [PubMed]

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