LYME MALADIE (Borréliose)

Une peste de l'ignorance ce qui concerne la méconnaissance d'une Peste

Mon objectif pour la rédaction de ce rapport est de contribuer à éclairer les profanes et de la communauté médicale au sujet de la situation extrêmement complexe de l'infection appelée la maladie de Lyme. J'ai récemment été infecté par la maladie de Lyme et je ferai part de mon expérience et de ce que j'ai appris sur la nature complexe de la maladie de Lyme dans le présent rapport.
Je continue de mettre à jour ce rapport et je prévois de nombreuses révisions à l'avenir, à mesure que plus d'informations concernant la maladie de Lyme les surfaces. J'ai récemment été mis en place pour une importante percée médicale découvert par le Dr Trevor Marshall, directeur de recherche à l'Autoimmunity Research Foundation en Thousand Oaks, CA. Nous avons mis à jour cette version du rapport d'inclure cette information essentielle.

La maladie de Lyme (LD) est un sérieux complexe multidisciplinaire maladie inflammatoire du système qui sera déclenchée par le lipoprotéines bactériennes (BLPs) produite par la spirale en forme de bactérie appelée Borrelia. Borrelia sont difficiles à isoler, les cultiver, et à l'étude en laboratoire. Alors, nos connaissances techniques de cet agent pathogène est faible par rapport à notre compréhension de la plupart des bactéries qui causent la maladie. La transmission de Borrelia se produit principalement par la piqûre de tiques. La maladie touche tous les tissus et de tous les principaux organes du système dans le corps.
Cliniquement, elle peut apparaître comme une chronique arthalgia (douleurs articulaires), fibromyalgie (fibreux du tissu conjonctif et des muscles), la fatigue chronique, le dysfonctionnement immunitaire et les maladies neurologiques. LD peut même être mortelle dans les cas graves.

Le diagnostic de la maladie de Lyme primaire est fondée sur des preuves cliniques. Il n'existe actuellement aucun test de laboratoire qui est définitive pour la maladie de Lyme. De nombreux essais de donner de faux résultats négatifs.
Les médecins ne connaissent pas bien la complexité de la présentation clinique de la maladie de Lyme fréquemment misdiagnose comme d'autres troubles tels que: fibromyalgie ou de fatigue chronique Immune Dysfunction Syndrome (CFIDS), la sclérose en plaques, Lupus, le Parkinson, la maladie d'Alzheimer, Arthrite rhumatoïde, Motor Neuron Disease (SLA, sclérose latérale amyotrophique - Lou Gherig la maladie), Multiple Chemical Sensitivity Syndrome (MCS) et de nombreux autres troubles psychiatriques comme la dépression et l'anxiété.

La maladie de Lyme est un nom familier pour la plupart des gens, mais leur connaissance de celui-ci est très limité. Malheureusement, cela est également vrai pour la plupart des professionnels de la communauté médicale. Il ya eu de nombreux rapports dans les médias à ce sujet aux États-Unis au cours des 25 dernières années. Ces articles relatent superficielles quelque chose au sujet de petits cerfs tiques transmettant bactérie *Borrelia burgdorferi.* Le cocher vecteurs sont censées être principalement limité à certaines zones endémiques des États-Unis, qui sont le Nord-Est et la partie supérieure du Midwest.
Fréquemment mentionné est la cible des éruptions cutanées qui se développe suite à la piqûre d'une tique infectée. La maladie est signalée à commencer par des symptômes grippaux que les progrès d'une arthritique et fibromyalgic état. On dit souvent que la maladie de Lyme peuvent être facilement traitées avec des schémas d'antibiotiques. Bien que ces rapports sont partiellement vrai, ils sont également très gravement erronées et fallacieuses!

Ce rapport est urgent d'avertissement pour tout le monde. La maladie de Lyme est dévastateur sur la vie de centaines de milliers de personnes, et nous sommes tous menacés. De nombreux patients souffrent de la maladie de Lyme chronique et continuent d'être mal diagnostiqués et maltraités. Dans de nombreux cas de la maladie de Lyme, un diagnostic correct ne se produit qu'après plusieurs mois ou plus souvent de nombreuses années de souffrance à la maladie. D'ici là, il a provoqué de graves maladies, d'invalidité et de dommages permanents. La maladie est répandue, et la prévalence est sensiblement plus élevé que rapportés par des responsables de la santé.

Il est très regrettable que la plupart des médecins ne savent pas comment reconnaître et traiter les cas de la maladie de Lyme, en particulier l'illusoire cas de la maladie de Lyme chronique. Je ne suis pas juste parler générales MDs étant ignorants; Je parle aussi de spécialistes tels que: rhumatologues, neurologues, chirurgiens orthopédistes, cardiologues, psychiatres, et les plus ignorants effectivement semblent être spécialistes des maladies infectieuses. J'ai été extrêmement surpris par ce fléau de l'ignorance une fois que j'ai commencé mon enquête de la maladie de Lyme.

Il existe certains facteurs clés qui existent dans la communauté médicale en ce qui concerne la maladie de Lyme; Ils vont un long chemin en expliquant pourquoi LD est souvent mal diagnostiqués et maltraités:

1. **LD est souvent mal diagnostiqués.**
   Les médecins souvent négliger les cas de la maladie de Lyme, tout simplement parce qu'ils ne connaissent pas la complexité de la pathogenèse de la maladie. Ils ne comprennent pas que la maladie de Lyme provoque bien plus de 100 symptômes différents; La commune arthralgies (terme médical pour douleurs articulaires) est un symptôme LD que la plupart des médecins connaissent; Toutefois, il n'est qu'un des nombreux symptômes causés par la maladie de Lyme .
   La présentation clinique de la maladie de Lyme peut être très subtile et complexe. La plupart des médecins ne savent pas que les tests de laboratoire sont souvent inutile et trompeuse. Les résultats sont négatifs ou peu fréquemment chez les individus présentant une borréliose. La technologie que nous avons affaire à borrelia espèce est dans le besoin d'amélioration significative. L'isolement et l'identification de borrelia est rarement réussi; Et aucun test de laboratoire clinique existe qui peuvent définitivement diagnostiquer la maladie de Lyme.
   C'est pourquoi un diagnostic de la maladie de Lyme est largement fondé sur les informations cliniques telles que l'histoire, les symptômes, et la réponse à la thérapie. Il est un art de la médecine lorsqu'il s'agit de la maladie de Lyme.
   Lyme connu un vif médecins doivent utiliser les compétences cliniques et de jugement lorsqu'il s'agit de suspecter la maladie de Lyme les patients.
   Ils soigneusement évaluer le patient et les symptômes de l'histoire lorsque la recherche d'un diagnostic et sont même en mesure de reconnaître les symptômes du complexe subtil Lyme cas. La plupart des médecins ne reconnaissent pas les symptômes de la maladie de Lyme chronique, et se reposer uniquement sur les tests de laboratoire pour confirmer un diagnostic. Souvent ces tests en laboratoire sont négatifs et d'induire en erreur le médecin et le patient à la recherche d'une autre cause. La plupart des médecins ne savent pas que borrelia produire une grande variété de bactéries toxiques lipoprotéines (BLPs) et ils ne sont pas familiarisés avec la façon dont ces BLPs provoquent des maladies. Spécialistes de la maladie de Lyme doivent utiliser très vive jugement clinique des cas lors du diagnostic de la maladie de Lyme. En défense de l'ignorance des médecins, beaucoup de reproches peuvent se reposer sur des responsables de la santé et les établissements médicaux qui ne sont pas cliniciens donnant les informations appropriées dont ils ont besoin pour ces cas difficiles diagnostic de la maladie de Lyme.
   Les critères utilisés pour rendre la maladie de Lyme par les médecins est souvent déterminé par l'état de santé des fonctionnaires et est souvent basé sur la rigidité des critères établis par les Centers for Disease Control and Prevention (CDC).
   Cette CDC critères a été établie pour une enquête épidémiologique, qui a été conçu pour étudier la répartition de la maladie de Lyme.
   La méthode en deux étapes de la CDC utilise une immunologique de dépistage pour tous les patients suivis d'une plus sensibles et spécifiques western que si le test de dépistage a été positive. Malheureusement, cette approche a été initialement prévu pour la surveillance de la maladie de Lyme en potentiellement les patients asymptomatiques, et non à des fins de diagnostic chez les patients présentant des symptômes qui sont potentiellement liées à la maladie de Lyme.
   Ce critère n'était pas destiné à être utilisé comme un standard pour le diagnostic clinique de la maladie de Lyme; CDC a clairement indiqué.
   Malheureusement, l'ignorance des responsables de la santé et les médecins continuent d'utiliser ces critères pour le diagnostic clinique de la maladie de Lyme.

2. **Pathogenèse connaissait pas.**
   La maladie de Lyme est complexe pathogenèse que je vais aborder dans la suite de ce rapport. Seul un petit nombre de professionnels de la santé à comprendre la pathogenèse de la maladie de Lyme. En fait, très peu de médecins qui se spécialisent dans la maladie de Lyme comprendre cette pathogenèse très bien. Cette information détaillée n'est pas enseignée dans les écoles de médecine, ou même dans la médecine générale, des conférences ou des résidents dans les pays qui sortent des séminaires. Ainsi, la plupart des cliniciens à pratiquer la médecine ne comprends pas comment borrelia provoque la maladie. Sans cette connaissance, il est bien difficile de reconnaître, de diagnostiquer et de traiter la maladie de Lyme.

3. **LD est provoqué par de nombreux borrelia espèces.** Une autre grande supervision de la communauté médicale en ce qui concerne la maladie de Lyme est que *Borrelia burgdorferi* n'est pas la seule bactérie qui cause la maladie de Lyme; Il existe de nombreux pathogènes borrelia souches; Dont beaucoup causer borréliose (comme la maladie de Lyme) . L'agent étiologique, *Borrelia burgdorferi,* est un type de spirochete. Quand *Bb* a été découverte pour la première fois en 1982, on pensait qu'il n'y avait qu'une seule souche. Depuis lors, environ 100 US et 300 monde souches de la bactérie ont été découvertes.

   Au milieu des années 1990 genospecies ont été constitués pour regrouper les nombreuses variations en

sous-catégories.

Borrelia burgdorferi est pris lato sensu, nom donné à l'ensemble de la catégorie. En Amérique du Nord, il n'ya qu'un seul genospecies variante - *Bb stricto sensu.* En Europe, il existe trois catégories Bb stricto *sensu,* B. *Garinii, et* B. *Afzelii.* L'Asie a *B. Garinii et* B. *Afzelii.* Le Japon a *B. Japonica et* B. *Miyamoto.* Ces groupes évoluent à mesure que de nouvelles découvertes se produisent.

Un nouvel agent pathogène provoquant Lyme ou "Lyme - comme" la maladie a été signalée. Bien qu'elle ne soit pas cultivables, il a été nommé *B. Lonestari* ps.

B. andersonii, B. Lonestari *et* B. *Miyamotoi* ont été identifiés par PCR et analyse de séquences d'ADN humain probable que les agents pathogènes dans le U. S. Malheureusement, les critères de Lyme cliniques sont fixés pour seulement *Borrelia burgdorferi;*
Ils n'ont pas été conçus pour toute autre borrelia espèces. La raison en est que *Borrelia burgdorferi* Suivis par les responsables de la santé, mais pas les autres espèces est dû au fait que c'est la première borrelia espèces que les laboratoires sont capables d'identifier et d'étude. J'avoue que Borrelia espèces sont très difficiles à cultiver (fastidieux) et le travail dans le laboratoire. Dans la plupart des cas, les laboratoires ne sont même pas en mesure d'isoler et d'identifier les espèces Borrelia. Parmi les autres souches connues de borrelia comprennent: *B. Valaisiana,*

B. lusitaniae *et* B. *Bissettii.*

4. **Il ya plusieurs transporteurs de LD que simplement cocher le cerf.** Il ya un énorme malentendu sur le vecteur (transporteur) qui transmet la maladie de Lyme. Tout d'abord, le familier cocher vecteur appelé le cerf tique (Ixodes dammini) et les tiques à pattes noires (communément appelé chevreuil tiques) (Ixodes scapularis) sont plus fréquentes et à la diffusion plus large que rapportée. Deuxièmement, ces tiques ne sont pas le seul vecteur capable de transmettre Borrelia espèces. Cocher plusieurs autres espèces telles que le Lone Star tiques (Ammblyoma americanum), dans l'ouest de tiques à pattes noires (Ixodes pacificus), le bois et les tiques ou chien tiques (Dermacentor Varié) peut transmettre aussi. Malheureusement, cette information cruciale n'est pas signalé par les responsables de la santé publique et à la communauté médicale. La généralisation de la distribution de ces vecteurs cocher accroît considérablement la prévalence de la maladie de Lyme bien au-delà de celle des rapports officiels. Le public doit comprendre le danger potentiel de toutes les tiques, et pas seulement celle du cerf cocher.
5. **LD est plus fréquent que nous le pensons.** La prévalence réelle de la maladie de Lyme est beaucoup plus élevé que ce qui est actuellement signalée par les responsables de la santé. Il est difficile de savoir combien de cas sont non déclarée, mais les estimations indiquent que la prévalence est effectivement 10-15 fois plus élevé que ce qui est effectivement d'être signalés. Personnellement, je pense qu'il est beaucoup plus que cela. Pourquoi les responsables de la santé sous notification des cas de la maladie de Lyme? Là encore, la réponse est parce que les médecins ne reconnaissent pas et de rendre la plupart des cas. Ces cas ne sont pas signalées objet d'un diagnostic erroné, même si la maladie de Lyme est une maladie à déclaration obligatoire (dans l'État de l'Iowa).
   Donc, un cycle de futiles existe causant de nombreux cas de la maladie de Lyme d'être mal diagnostiqués et non déclarée.
   Telle est, depuis la plupart des cas de la maladie de Lyme vont pas diagnostiquée, les responsables de la santé au titre de la maladie de Lyme - rapport; Ainsi, les médecins qui lisent leurs rapports officiels estiment que la prévalence de Lyme est rare et le placer bas sur leur liste de possibilités face à des cas cliniques Qui pourrait être causée par Borrelia.
6. **Les patients doivent, plus longue et plus complète de traitement.** Le traitement standard de 4 -6 semaines de traitement antibiotique n'est pas suffisante pour traiter la maladie de Lyme chronique. Chronique de la maladie de Lyme est souvent une question de vie ou de longue maladie. Des mois, des années, et souvent indéterminée traitement antibiotique peut être nécessaire pour gérer la maladie. Ignorant les médecins ont souvent recours à la norme de traitement et de considérer le patient débarrassé de la maladie de Lyme après. Souvent, ces patients ne sont pas traités assez longtemps pour effacer le Borrelia obstinée de l'organisme. Ainsi, quand la norme régime d'antibiotiques est terminée, les malades de Lyme symptômes peu après la résiduelle Borrelia reemerges.
   Malheureusement, la rechute n'est pas souvent reconnu par les médecins et les patients sont mal diagnostiqués avec un autre désordre.
   Non seulement le traitement sera réalisé à l'infection; Il doit également gérer l'inflammation, de contribuer à éliminer les BLPs produit, le soutien du système immunitaire, et de nombreux autres problèmes connexes tels que les déficits hormonaux.
7. **Un diagnostic erroné conduit à mauvais traitements.** Un autre point critique qui mérite d'être soulignée est

celle de Lyme ignorent souvent les médecins qui administrent les médicaments est contre-indiquée chez les patients atteints de la maladie de Lyme.
Le traitement le plus souvent prescrit qui est extrêmement contre-indiqué est l'utilisation d'anti-inflammatoires stéroïdiens; Généralement les glucocorticoïdes (prednisone). Lyme patients souffrent de nombreux symptômes inflammatoires douloureuses.
MDs, ne sachant pas que le patient a la maladie de Lyme, pense qu'il est approprié de traiter ces patients avec les stéroïdes pour réduire la douleur et l'inflammation. Malheureusement, *la thérapie stéroïdiens est très néfastes pour les patients atteints de Lyme parce qu'elle supprime le système immunitaire du patient et la faire tolérer la présence de Borrelia au lieu d'attaquer et de tuer.* Ce dangereux traitement diminue significativement le pronostic des patients atteints de Lyme; Elle prolonge le cours de La maladie et la rend plus graves à long terme.

Lyme Wise Medical Doctors: The Few, le avertis, le harcelés

Le fléau de l'ignorance qui entoure la maladie de Lyme rend très controversée au sein de la communauté médicale. La plupart des médecins sont ignorants à propos de la nature complexe de la maladie de Lyme et sont souvent irrités lorsqu'ils sont confrontés à ce sujet.
Il n'ya que quelques médecins dans le pays qui sont bien informés sur la maladie de Lyme; Ils sont souvent appelés Lyme alphabétisés MDs (LLMDs) par le public et consciente de Lyme par leurs patients atteints de Lyme. La plupart LLMDs savoir sur la maladie de Lyme parce qu'ils l'ont étudié indépendamment. Le MD formelle de la formation à l'école de médecine et de la communauté médicale établie en ce qui concerne la maladie de Lyme est maigre.

LLMDs ont été et continuent d'être harcelés par la communauté médicale, par des responsables de la santé, par leurs pairs et collègues, les conseils médicaux par l'Etat et par les compagnies d'assurance pour le diagnostic et le traitement des patients Lyme au-delà des normes fixées par l'établissement. Malheureusement, certains de ces LLMDs ont interrompu le traitement des patients atteints de Lyme due au harcèlement. Quelques LLMDs ont effectivement eu leur licence révoquée médicale parce qu'ils ont traité les patients Lyme au-delà des normes fixées par la communauté médicale.
Par exemple, le traitement des patients avec les antibiotiques pendant plus de la norme 4.6 semaine laps de temps peut conduire à des harcèlements.

Si Lyme patients sont assez chanceux pour trouver une LLMD et obtenir le bon diagnostic et de thérapie, devinez quoi?
Beaucoup de compagnies d'assurance maladie sont en baisse de payer pour le traitement antibiotique approprié.
Pourquoi?
Si ce n'est pas le standard établi par la communauté médicale ignorants, les compagnies d'assurance ne veulent pas payer.
Le traitement standard pour la maladie de Lyme, qui est considéré comme un 4.6 semaine d'antibiotiques, est trop courte pour la plupart des cas de Lyme.
Un traitement de base pour la maladie de Lyme chronique nécessite habituellement au moins de 8 à 16 mois d'antibiotiques appropriés.

Tout au long de mes études de classique et à la médecine alternative, je rencontre souvent les insuffisances de la médecine conventionnelle.
Leur ignorance qui entoure la maladie de Lyme (LD) peut être l'un des plus grands échecs médicaments conventionnels.
En fait, je le placer là avec leurs conséquences désastreuses faible en gras, faible en calories alimentation recommandation.
Je suis extrêmement déçu par le peu qu'ils savent et le peu qu'elles ne concernant la maladie de Lyme. Cette ignorance ne devrait pas exister. J'ai pu me former sur la maladie de Lyme. Il ya de nombreuses bonnes ressources sur l'Internet qui sont pleins d'informations utiles concernant la maladie de Lyme. J'ai mis quelques liens sur LD sur mon site qui se trouve à: www.myremedi.com.
L'établissement médical doit relier le complexe tableau clinique de la maladie de Lyme avec les éléments déjà présents dans la littérature scientifique.
Je sais qu'ils ne tiendra pas compte des preuves anecdotiques qui est très utile dans la compréhension de la maladie de Lyme cliniquement, mais n'est pas considérée comme scientifique. Malheureusement, il n'ya pas assez mitigés dans l'art cliniques médecine conventionnelle.

Les cliniciens ont besoin de ralentir et d'écouter attentivement leurs patients; Le patient sera généralement leur dire ce qui ne va pas.

Médecin doit utiliser les compétences cliniques de leur mieux pour écarter la maladie de Lyme lorsque l'on traite avec un patient présentant des symptômes évoquant complexe de la maladie de Lyme. Ils doivent éduquer themselve sujet de la maladie de Lyme.
Le CDC critères n'est pas une béquille pour eux à utiliser pour faire le diagnostic clinique.

L'histoire de la maladie de Lyme

La maladie de Lyme est relativement jeune dans l'histoire U. S. Le premier cas signalé borréliose aux Etats-Unis a été faite par le Dr Rudolph Scrimenti en 1970.
Il a remarqué une lésion cutanée appelée érythème migrants (EM), une éruption que Borrelia peut provoquer rapidement après une tique mordant.
Scrimenti diagnostic et le traitement du patient qui avait été mordu par une tique et LD acquise lors de la chasse dans le Wisconsin.
La maladie de Lyme a été retrouvée plus tard dans la célèbre Lyme, Connecticut flambée qui a commencé en 1975 par le Dr Allen Steere.
Il a mal diagnostiqués cette flambée comme l'arthrite rhumatoïde juvénile près de la communauté de Lyme, dans le Connecticut. À cette époque, la condition a été nommé 'Lyme l'arthrite. 'Puis, au début des années *1980,* B. *Burgdorferi*
a été reconnu comme l'agent étiologique de l'épidémie de Lyme et le nom a plus tard été changé pour la maladie de Lyme.

Bien que la maladie a été nommé pour la ville dans le Connecticut, la maladie de Lyme (borréliose) effectivement a une longue histoire en prenant elle aussi loin que les années 1800. Le premier cas signalé de la maladie de Lyme clinique a été faite en Allemagne par le docteur Alfred Buchwald en 1883. Il a décrit une maladie de la peau dégénérative maintenant appelé acrodermatites chroniques atrophicans (ACA), qui est connue pour être provoquée par Borrelia.
Ainsi, un grand nombre d'informations déjà connues sur la maladie de Lyme avant sa redécouverte comme la maladie de Lyme aux États-Unis.
Toutefois, la plupart de ce début de l'histoire médicale de la DL européenne vient de la littérature scientifique. Les Etats-Unis reste à la traîne en ce qui concerne les Européens, leur compréhension de la borréliose.

La terminologie employée pour décrire Borrelia infections et la maladie causée par eux n'est pas clairement défini. Le nom de "maladie de Lyme" a été utilisé pour décrire la maladie spécifiquement provoquée par B. burgdorferi *seulement, et non* d'autres espèces de Borrelia.
Cependant, le terme de la maladie de Lyme ou borréliose de Lyme est couramment utilisé pour décrire les maladies infectieuses qui sont causées par de nombreuses espèces de Borrelia. Plus appropriée le nom de la maladie causée par borrelia infections est *borréliose.*
Toutefois, borréliose n'est pas couramment utilisé et n'est pas familier pour la plupart, donc je vais utiliser le plus souvent familier nom, la maladie de Lyme.
Cependant, veuillez noter que je vais utiliser borréliose, maladie de Lyme, ou borréliose de Lyme pour décrire la complexité de la maladie causée par borrelia les infections.

Le Spirochete appelé Borrelia: Qu'est-ce qui fait que la maladie de Lyme Tick

Borrelia espèces, à l'instar de l'homme pathogène *Treponema pallidal,* (la cause de la syphilis) sont placés dans le spirochete famille de bactéries.
Spirochetes sont longues, minces, en forme de spirale bactéries qui ont flagelles (queues), *(voir* les figures *1 et 2 ci-dessous).*
Autres familiers humains maladies provoquées par Borrelia espèces comprennent: 1) la fièvre récurrente, qui utilisent les tiques molles ou des poux, comme des vecteurs, et 2) les maladies de gencive. Le genre Brachyspira est un spirochete que provoque dans la colite porcine en Europe et a été récemment découvert à causer la colite chez l'homme.

Spirochetes sont très difficiles à cultiver (fastidieux), d'identifier et d'étudier en laboratoire. Dans le laboratoire nous ne pouvons pas encore croître le spirochete qui cause la syphilis, *Treponema pallidal.* Cela rend très difficile d'effectuer des recherches et mettre au point de meilleurs tests de diagnostic et des thérapies de la borréliose.
Borrelia espèces poussent très lentement; Ils font même pousser beaucoup plus lent que les champignons et les mycobactéries. Leur taux de croissance est également plus lente que *de Mycobacterium tuberculosis,* qui cause la tuberculose.
Borrelia de la lenteur de la croissance explique en partie sa capacité à causer des maladies chroniques et de la difficulté d'identifier dans le laboratoire.

Schematic representation of a spirochete

Borrelia existe trois formes de vie différentes: 1) le kyste, 2) la spheroplast ou "L forme", qui n'a pas de paroi cellulaire (communément appelée: paroi de la cellule déficiente (CWD)), et 3) la spirale typique Bactéries en forme de formulaire qui a une paroi de la cellule et des flagelles comme on le voit dans les figures 1 et 2. Spirochetes ont un mode de motilité qui lui permet de se déplacer facilement à travers les tissus de l'organisme. Par la rotation de leurs filaments axiaux (endoflagella) le flagelle tourne provoquant la spirochete à aller dans un bouchon à vis de la mode.
Ce mode de motilité permet spirochetes à littéralement "vis" en eux-mêmes et à travers les tissus de l'organisme. Ils peuvent également comme un contrat de printemps et de se déplacer dans les tissus comme ils uncoil. Spirochetes cacher leurs flagelles de l'hôte de défenses immunitaires, qui sont normalement antigéniques et déclencherait une réponse immunitaire si détecté.

Figure 1

En 1982, le brillant entomologiste, Willy Burgdorfer, a découvert les agents étiologiques de la maladie de Lyme. Il enquêtait sur les tiques à la United States Rocky Mountain Laboratories située dans le Montana, qui fait partie du National Institutes of Health, lorsqu'il a isolé spirochetes partir de la mi - cran *d'Ixodes* tiques. Il a montré que ces spirochetes réagi avec sérum de patients qui avaient reçu un diagnostic de la maladie de Lyme.

En conséquence, les agents étiologiques a été nommé en son honneur, *Borrelia burgdorferi (Bb).* Il est intéressant de noter que le Dr Burgdorfer connaissait bien la littérature européenne concernant borréliose et a été en mesure de relier cette information à la flambée de Lyme.
Je souhaite que le reste de notre établissement médical serait aussi diligente que lui.

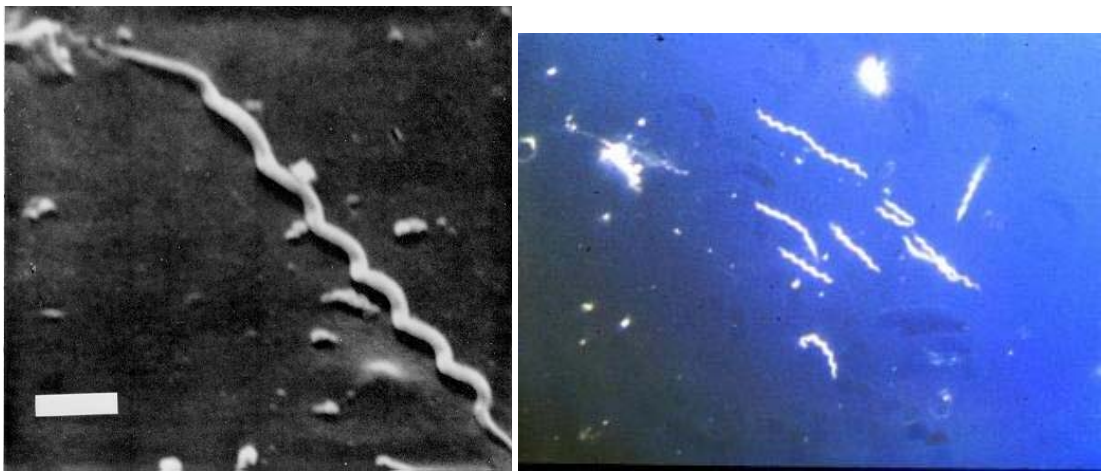


Figure 2: *Borrelia burgdorferi,*
la bactérie qui cause la maladie de Lyme, photo de gauche vu à 400x de grossissement. Est une bonne image, la microscopie électronique à balayage de *Treponema pallidal,* la cause de la syphilis.

Arachnophobia justifiée: tiques, le vecteur de Borrelia

Tiques sont des parasites externes sang qui se nourrissent de l'homme, les mammifères sauvages et domestiques, et les oiseaux.
Les tiques ne sont pas les insectes; Ils sont 8 pattes avec chiques arachnides, les araignées et des acariens.
L'épidémie de la maladie de Lyme Lyme dans le Connecticut retour en 1975 s'est produite dans un cadre champêtre et de l'apparition de la maladie au cours de l'été et au début de l'automne indiqué qu'un arthropode vecteur était probablement responsable de la transmission de la maladie. Comme mentionné plus haut, la principale méthode de transmission de dinars a été découvert grâce à tiques.

Je tiens à souligner de nouveau qu'il ya un énorme malentendu sur le vecteur (transporteur) qui transmet la maladie de Lyme.
Encore une fois, le familier cocher vecteurs appelé le cerf tique (Ixodes dammini) et à pattes noires cocher (aussi

communément appelé chevreuil tiques) (Ixodes scapularis) sont plus fréquentes et plus vastes que rapportées. Ces tiques ne sont pas le seul vecteur capable de transmettre Borrelia espèces. Plusieurs autres espèces dont le cocher Lone Star tiques (Ammblyoma americanum), dans l'ouest de tiques à pattes noires (Ixodes pacificus), le bois tiques ou American chien tiques (Dermacentor Varié) et le chien de cocher Brown (chiens sont les seuls hôtes de ces tiques) peut Le transmettre, aussi.
Malheureusement, les responsables de la santé ne sont pas signalé cette information cruciale, et donc, le public et la communauté médicale ne sont pas informés à ce sujet.

Depuis la large diffusion de ces vecteurs cocher accroît considérablement la prévalence de la maladie de Lyme bien au-delà de celle des rapports officiels.
Le public doit comprendre le danger potentiel de toutes les tiques, et pas seulement celle du cerf cocher.

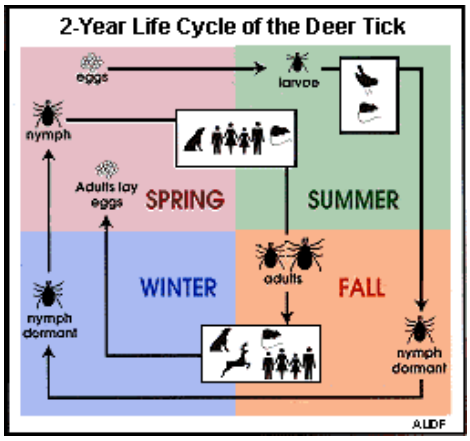


Figure 3

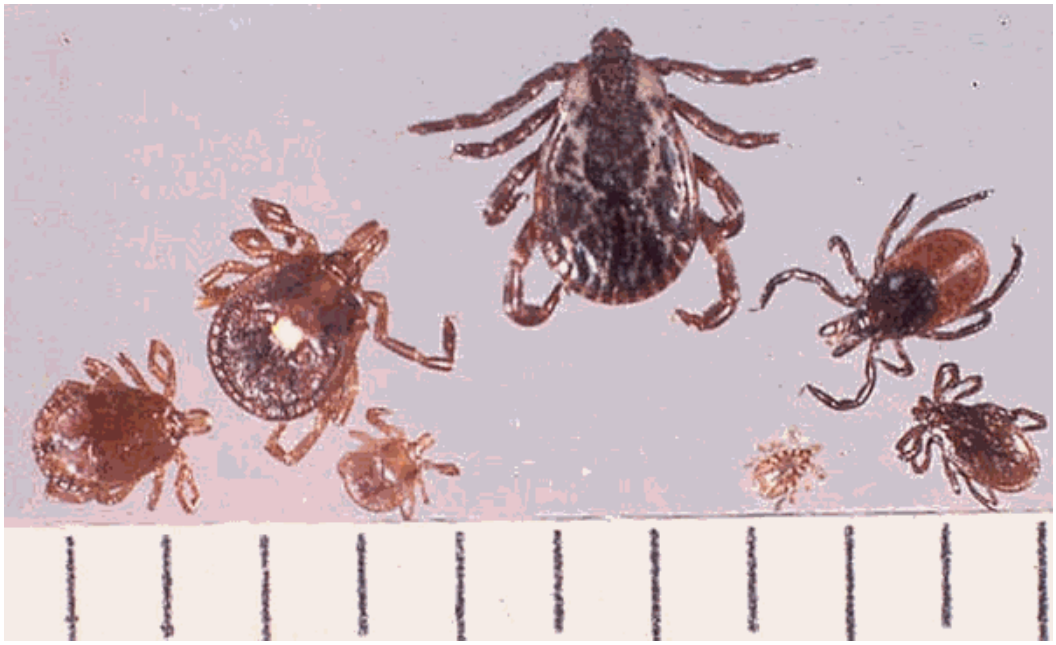
Le cycle de vie des tiques Les tiques ont un cycle de vie de 2 ans, qui comprend l'œuf, larve, nymphe, et adulte étapes *(voir* la figure *3)*.
Les nymphes et les adultes semblent être le principal responsable de la transmission de la borrelia d'hôtes sensibles grands animaux y compris les êtres humains; Cependant, les larves peuvent transmettre borrelia à l'homme également.

Au printemps, les œufs éclosent en larves ("semence" cocher), qui ont six pattes. Les larves se nourrissent et mue en nymphe qui possèdent huit jambes et pas de différenciation sexuelle. Nymphe mue en aliments pour animaux et des adultes qui sont différenciés en mâle ou femelle. Semblable au moustique, la tique femelle adulte a besoin d'un repas de sang avant de pouvoir pondre leurs œufs.

Un cocher cherche un repas de sang au niveau ou légèrement au-dessus du niveau du sol par l'escalade sur la végétation et l'aide de ses pattes et de se sentir récupérer un hôte. Ils sont habituellement trouvés entre sol à trois pieds au dessus du sol.
Pour trouver un hôte, une tique détecte le dioxyde de carbone, l'odeur, la chaleur corporelle, et, éventuellement, d'autres des stimuli générés à partir de l'hôte du corps. Tiques généralement nourrir une fois au cours du printemps, mais ils peuvent nourrir à l'automne et à tout moment durant l'année. Ils sont actifs à des températures supérieures à 42 ° F.
Bien que l'incidence de Borrelia induite erythra migrants (EM) irritations de la peau est la plus élevée au cours du printemps et au début de l'été, les symptômes de LD peuvent se produire à tout moment durant l'année.

Tiques ne peuvent pas survivre à une longue exposition au soleil et sont donc l'on retrouve habituellement dans les zones ombrées.
Cochez les habitats doivent contenir à la fois des petits animaux hôtes pour cocher larves et des grands animaux hôtes pour cocher les adultes.
Une humidité relative supérieure à 65% est nécessaire pour l'éclosion des oeufs et larves de survie. Ce frais et pluvieux, le printemps de 2003 a été idéal pour la production de tiques. Cela signifie qu'il y aura une grande récolte de nourrir les nymphes cet automne et le printemps prochain; Suivie d'une grande culture de l'alimentation des adultes tout au long de l'année 2004.

Un préféré cocher habitat est la zone de transition entre le bois et les pelouses ou prairies. Tiques devenir stimulé par la chaleur et le dioxyde de carbone qui est produit par une foule qui consacre un temps considérable dans les tiques' environnement. Tiques grippent le passage d'un hôte ou tombent au sol, de trouver l'hôte, et de monter sur elle. Ils se déplaceront vers le haut jusqu'à ce qu'ils atteignent la tête ou qui sont masqués. ***Il est important de rappeler que les êtres humains peuvent être les hôtes de tous les stades de tiques.***



Le cerf tique est de la taille d'un pinhead au stade de larves. Il est bronzage, et se nourrit de petits animaux comme la souris où il peut reprendre le spirochete. Au cours de la nymphe stade de la tique est de la taille d'une graine de pavot. Il est le beige ou partiellement transparent et se nourrit de plus gros animaux tels que des chats, des chiens et des humains.
Adulte cerfs tiques sont en noir et / ou rougeâtre et se nourrissent des bovins, les cerfs, les chiens et les humains.

Figure 4: Gauche est la *Amblyomma americana »(Lone* Star cocher) nymphe, adultes et larves. Centre est le *Dermacentor Varié* (American chien ou bois cocher) adulte. Droit est le *Ixodes scapularis* (tique à pattes noires) larve, adulte, et nymphe.

Figure 5: Lone Star tique, *Amblyomma americanum*

Le Prêt Star tiques obtenir leur nom commun de la seule tache argentée située sur le dos de la femme *(voir figure 5).* Lone étoile tiques attaquent les humains plus fréquemment que n'importe quel autre cocher dans l'est et le sud-est du pays. Sa distribution a lieu dans le centre-ouest du Texas vers le nord, jusqu'à l'Iowa et l'Est vers la côte atlantique. Il est particulièrement abondant dans la région et orientale Ozark Oklahoma. Des données récentes indiquent que le Lone Star cocher peut jouer un rôle important en tant que vecteur de Borrelia espèces, eg, Borrelia lonestari, dans le Sud et le Midwest.
Malheureusement, les responsables de la santé ne sont pas alerter le public sur cette grave menace. Les plus fréquents de tous les tiques dans le Midwest est le bois de cocher ou chien de cocher.

Figure 6:

Comparaison de la Capitulum (tête / bouche) de l'étoile solitaire tiques *(Amblyomma americanum)*, qui a manifestement une autre taille et la forme que celui de l'American cocher chien *(Dermacentor Varié).*

Ci-dessous - Magnified buccales de la tique cerf (I. dammini)

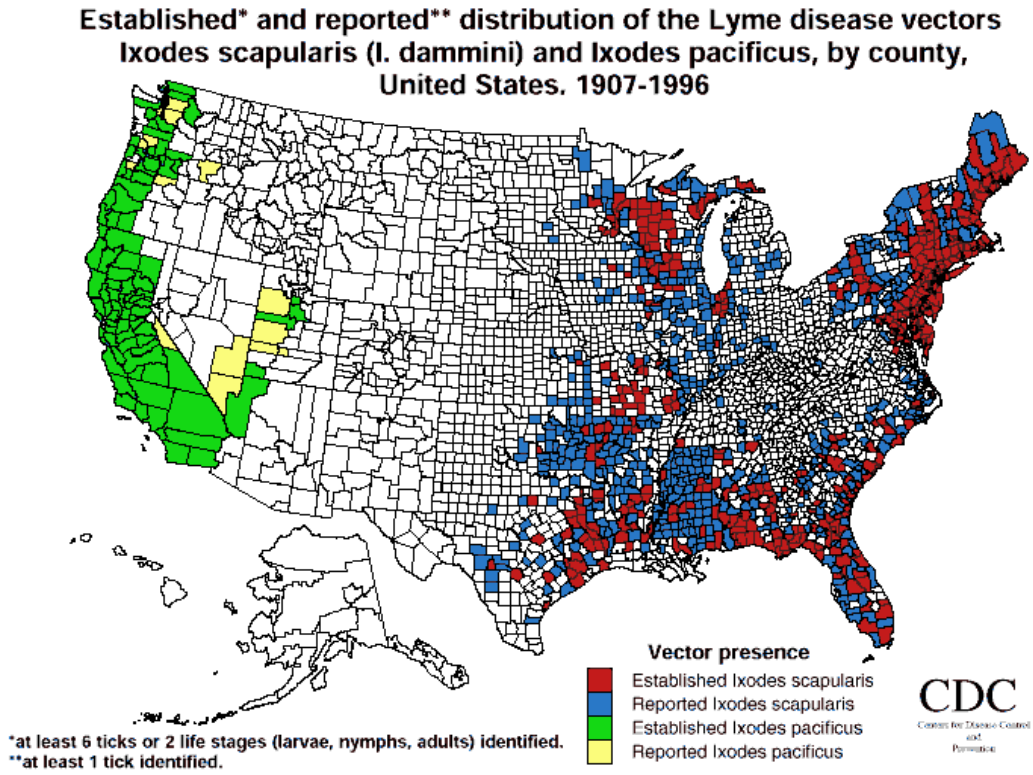


Figure 7

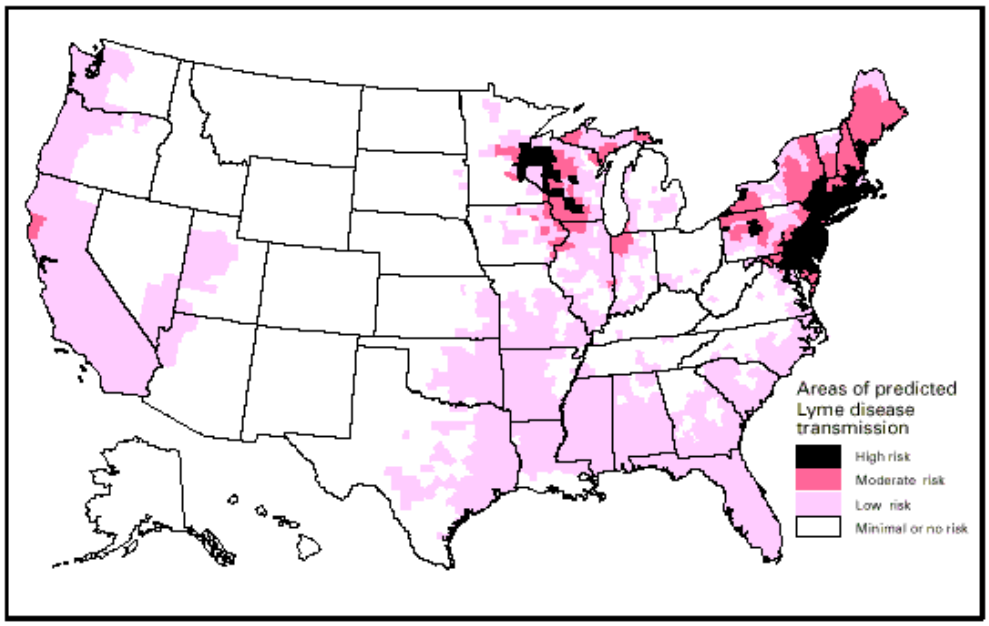
Figure 7 est un CDC tableau montrant la répartition des cerfs *(Ixodes scapularis)* et de l'ouest à pattes noires *(Ixodes pacificus)* tiques.
Malheureusement, ce tableau ne montre pas la grande distribution de la Lone Star cocher, American Dog Tick, Black unijambiste Tick cocher et d'autres vecteurs de Borrelia. Toujours selon la carence de rapports par le CDC, il ya trois points essentiels dans la moitié est du pays *(voir* la figure *8).*

Tiques sont transportées par un certain nombre de différents hôtes animaux, et non pas simplement des chevreuils. Les hôtes pour les tiques adultes sont nombreux et comprennent: le renard, le coyote, chiens, chats, bovins, le lapin, moufette, raton laveur, rat, écureuil, le cerf de Virginie, dindon sauvage, et les êtres humains. Nymphes obtenir sur un grand nombre de ces animaux, ainsi que les plus gros animaux typiques pour les adultes. Motif oiseaux nicheurs, y compris bobwhite cailles, faisans, dindes et poulets sont également cocher hôtes. Quarante-neuf espèces d'oiseaux migrateurs ont été trouvés à tiques, les transporter de grandes distances et en contribuant à la propagation de la maladie de Lyme.
Mineures vecteurs potentiels de Borrelia espèces comprennent: chiques, les moustiques, les mouches piqueuses et puces.
LD cas d'un contrat en cours de animaux infectés sont bien documentés; Probablement due à l'animal apportant des tiques infectées dans le foyer.
Borrelia peuvent également être transmis dans l'utérus d'une mère infectée à l'enfant pendant la grossesse. Congénitale peut provoquer la transmission de fausses couches, de troubles neurologiques graves handicaps, ou d'autres dysfonctionnements majeurs du système pour le bébé. Autres modes de transmission existent probablement que nous ne sommes pas au courant.
Je suis personnellement préoccupé par le fait que la transmission peut se produire lors d'une transfusion sanguine autorités sans le savoir.

Maladie de Lyme dans le U.S.A.

Même rapports épidémiologiques fondées sur des critères CDC, qui sous-estiment nettement le véritable clinique prévalence de 10-15 fois, montrent que la maladie de Lyme a augmenté de façon spectaculaire au point que la maladie est devenue un important problème de santé publique aux États-Unis. La maladie de Lyme est la plus répandue des maladies à tiques dans le U. S. LD cas ont été signalés dans l'ensemble de la partie continentale des États-Unis et sur plusieurs continents.

National Lyme disease risk map with four categories of risk



Note: This map demonstrates an approximate distribution of predicted Lyme disease risk in the United States. The true relative risk in any given county compared with other counties might differ from that shown here and might change from year to year. Risk categories are defined in the accompanying text. Information on risk distribution within states and counties is best obtained from state and local public health authorities.

Figure 8

En regardant cette carte, il est intéressant de souligner certains points:

- Le Nord-Est est le lieu traditionnel où vous pensez de Lyme et est tout à fait évident dans la figure 8.
- Le Midwest est un domaine en pleine émergence de Lyme activité est malheureusement peu et mal reconnus dans le présent plan. **Dr**
  Masters, un expert en Lyme Cape Girardeau, a montré que Borrelia est la cause la maladie de Lyme dans le Missouri.
- The South; from Texas to Florida, and from Georgia to Virginia, where knowledge of the disease is still very limited, tick populations are large, and vector-borne disease cycles are still poorly understood and under reported.

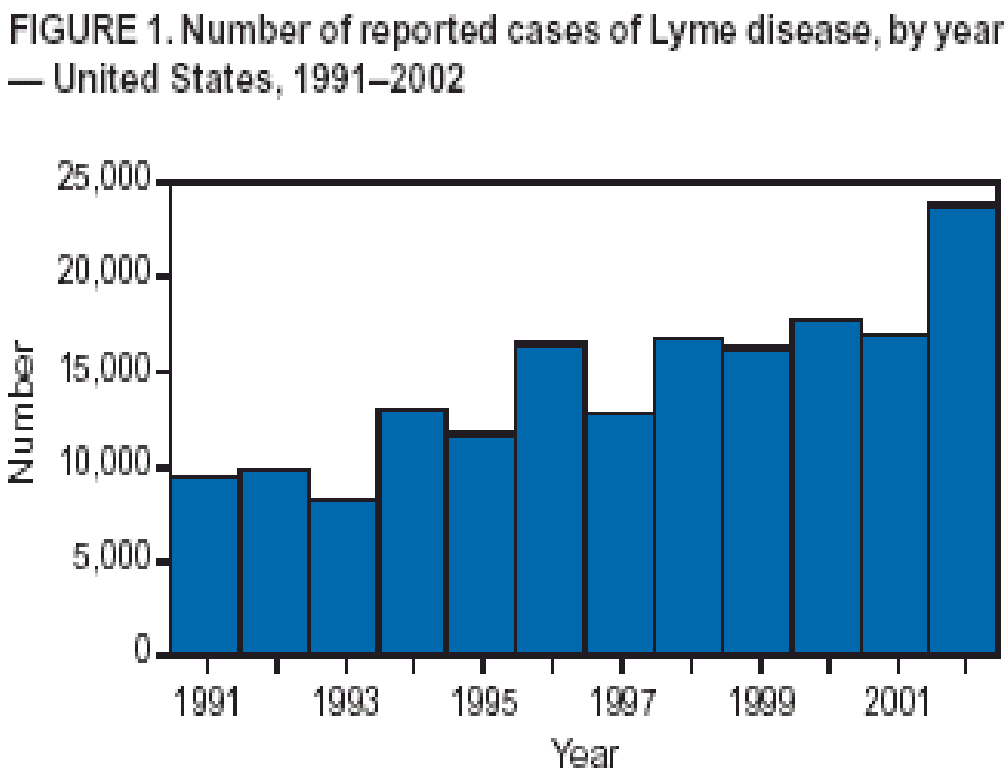


Figure 9

In 1996, there were 16,455 new cases reported in the U.S. The number dropped in 1997(see table above), but in 1998 there were 16,801 cases, and in 1999 there were 16,273 new cases reported to the CDC. In 2002, 18,000 cases of Lyme disease were reported in the U.S.

Reported cases of Lyme disease in 2002 were up nearly 40 percent from 2001, according to the CDC. The phenomena does not appear to be regional, as 95 percent of reporting states showed an increase in reported cases, with a one year doubling seen in some states. The northeastern and northcentral states remain a focus of concern, accounting for approximately 90 percent of all cases.

With 23,000 new cases of Lyme reported last year, it is becoming apparent that the disease is a much larger problem than many had originally thought. CDC statements acknowledge the prevalence of Lyme disease.

The Iowa Department of Public Health criteria for Lyme disease is actually much more restrictive than the CDCs criteria. A Lyme disease case must have three out of these five criteria:

- Live in an endemic area (Iowa is not listed as an endemic area)
- Known tick exposure
- A bulls-eye EM rash
- Early symptoms of Lyme
- Laboratory confirmation

Since 1982, 220 cases that fit the above criteria have been reported to the Iowa Department of Public Health. Twenty-six cases were reported in 1998. Wisconsin (5,970 cases since 1982), Minnesota (2,385 cases since 1982), and Missouri (1,060 cases since 1982) are all CDC endemic areas for Lyme disease.

Although Lyme is a mandatory reportable disease, physicians will often treat patients with early Lyme without getting positive serology tests and avoid reporting it to the Dept. of Public Health.

Pathogenesis of borreliosis: How Borrelia Cause

a Self-Perpetuating Inflammatory Disease

Understanding how borrelia causes disease is the key to successful diagnosis and treatment.

Bacterial Lipoproteins (BLPs)

Regarding borreliosis, the molecular component of the pathogen that appears initiate the pathogensis are the bacterial lipoproteins (BLPs) which are found within the outer surface proteins of the borrelia cell membrane.

BLPs are fat-soluble toxins that are part protein and part lipid. They are often a structural part of the borrelia cell membrane and can be found within the outer surface proteins of borrelia. They are very potent immunomodulators even in small amounts. Thus, a few borrelia can produce enough BLPs to initiate significant disease.

These BLPs trigger many harmful responses in any tissues and organ system of the human body. These responses, produce complex symptoms of fibromyalgia, arthritis, neurological signs, psychiatric disorders, immunologic dysfunctions, and endocrine deficiencies.

At the molecular level, the BLPs cause a dysfunction in the immune system by triggering a complex imbalance of chemical immune mediators (cytokines). These cytokines regulate the immune system and when they are over stimulated, they produce harmful reactions from the immune system, such as pain, inflammation, and even apoptosis (cell death). Some of the cytokines involve
d include: tumor necrosis factor-alpha (TNF-α), interleukins-6 (IL-6), fatty acid products (eicosanoids such as inflammatory prostaglandins, thromboxanes, and leukotrienes) that have potent inflammatory/physiological properties and many other cytokines pl ay a role in the pathogenesis of borreliosis.

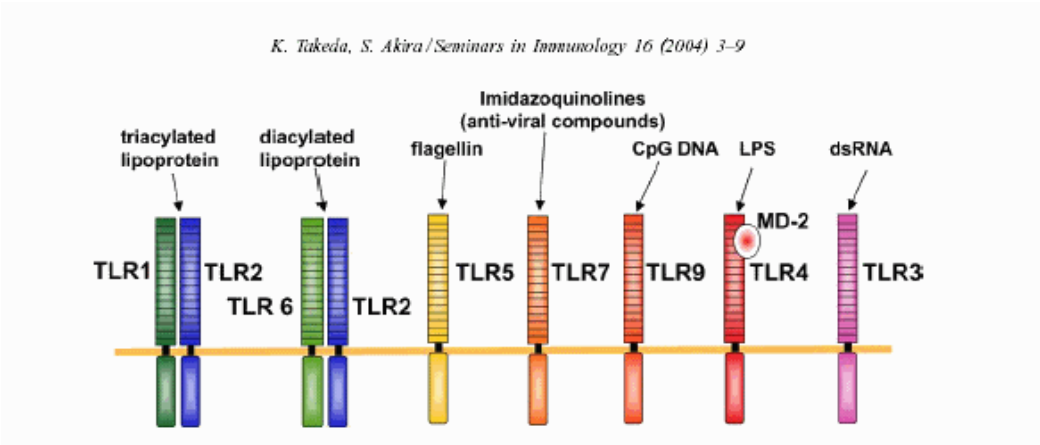
These BLPs have a key component, Pam3cys, which triggers an innate immune response that cascades into the disease borreliosis.

The inflammation triggered by the fat-soluble BLPs toxins is responsible for most, if not all symptoms of borreliosis.

So, the innate immune system and the acquired immune system are strongly triggered by the presence of the borrelia BLPs.

Toll-like Receptors and Innate Immunity

BLPs activates the innate immune system through what are called Toll-like receptors. Many cell types throughout the body carry the TLR receptors. It's a basic innate immune response that even invertebrates have. These receptors are able to recognize molecular patterns that are unique to microbial pathogens. The body uses TLRs to detect the prescence of many microbial pathogens, not just borrelia. The figure below shows how TLRs can detect several molecular patterns of different microbial pathogens. The TLRs that are most likely involved in borreliosis are TLR 2, 6, & 1, but TLR-2 likely plays the major role.



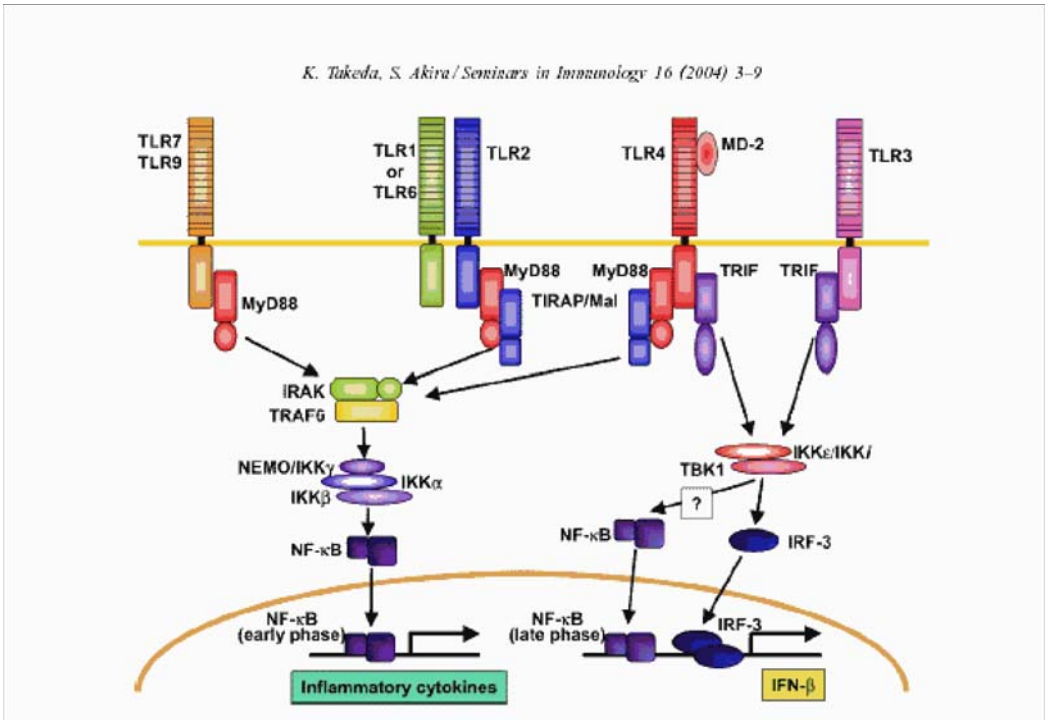
For a thorough review of the TLR signaling pathway see this reference: Seminars in Immunology 16 (2004) 3–9 ,TLR signaling pathways, by Kiyoshi Takeda, Shizuo Akira . Here's the abstract from this manuscript:

Abstract

Toll-like receptors (TLRs) have been established to play an essential role in the activation of innate immunity by recognizing specific patterns of microbial components. TLR signaling pathways arise from intracytoplasmic TIR domains, which are conserved among all TLRs. Recent accumulating evidence has demonstrated that TIR domain-containing adaptors, such as MyD88,

TIRAP, and TRIF, modulate TLR signaling pathways. MyD88 is essential for the induction of inflammatory cytokines triggered by all TLRs. TIRAP is specifically involved in the MyD88-dependent pathway via TLR2 and TLR4, whereas TRIF is implicated in the TLR3- and TLR4-mediated MyD88-independent pathway. Thus, TIR domain-containing adaptors provide specificity of TLR signaling.

We know that BLPs are potent activators of Toll-like receptor-2 (TLR2). Thus, through TLR2, BLPs induces the synthesis of the precursor of the pro-inflammatory cytokine interleukin -1B (IL-1B). As shown in the figure below, TLRs can activate a transcription factor known as NF-kappa B which stimulates the gene expression for inflammatory cytokines.



BLPs also activates caspase 1 and potentiates apoptosis (programmed cell death) via this route.

The lipid moiety of the BLPs contains a part that is responsible for triggering the TLRs. A synthetic analog of this moiety is called: tripalmitoyl-S-glyceryl-Cys-Ser-Lys4-OH (Pam3Cys).

Other important inflammatory mediators triggered by BLPs in immune cells are tumor necrosis factor-alpha (TNF-α) , IL-6, I L-12, INF-γ , and nitric oxide (NO).

A Major Medical Breakthrough; Dr. Trevor Marshall's Angiotensin Discovery

The following discovery by Dr. Marshall shows how a Th1 inflammatory response can become self-perpetuating via the following biochemical pathway. Please note that sarcoidosis has a very similar pathogenesis to borreliosis. Borreliosis may actually cause many cases of sarcoidosis.

The Angiotensin Hypothesis

New Treatments Emerge as Sarcoidosis Yields Up its Secrets

Authors:

Trevor G. Marshall, Ph.D.,

SarcInfo, Thousand Oaks, California, trevor.m@yarcrip.com

Frances E.(Liz) Marshall, Grad. Dipl. Pharm,

Los Robles Regional Medical Center, Thousand Oaks, California,
liz.m@yarcrip.com

We have concluded that sunlight fuels the inflammation of sarcoidosis, via 1,25-D and Angiotensin II, in the following manner:

> As the circulating concentration of 1,25-D increases within the inflamed tissue, a much larger quantity of hematopoetic stem cells differentiate to produce monocytes . Monocyte differentiation into macrophages and epithelioid giant cells is enhanced. The differentiating macrophages and giant cells release Angiotensin Converting Enzyme. This ACE catalyzes Angiotensin I to form Angiotensin II (A-II). The A-II then binds at A-II Type 1 receptors on the macrophages and activated T-lymphocytes , stimulating Nuclear Factor-kappaB (NF- κ B) to signal the release for a cascade of Th1 cytokines . At least one of these cytokines, Gamma Interferon, increases the amount of 25-D being converted to 1,25-D in the macrophages, which in turn catalyses the differentiation of monocytes into even more macrophages and giant cells.

Normally this inflammatory cycle is self-limiting, but, in the case of sarcoid patients, 1,25-D levels are poorly controlled, leading to upregulated production of monocytes, and their upregulated differentiation into the macrophages and epithelioid giant cells characteristic of sarcoid granuloma .

Reichel, et al, demonstrated that lipopolysaccharide from gram-negative bacteria stimulated the generation of 1,25-D within pulmonary alveolar macrophages from sarcoid patients. A bacterial pathogenesis is therefore consistent with the initial increase in paracrine 1,25-D concentrations needed to trigger the run-away inflammatory biochemistry described above.

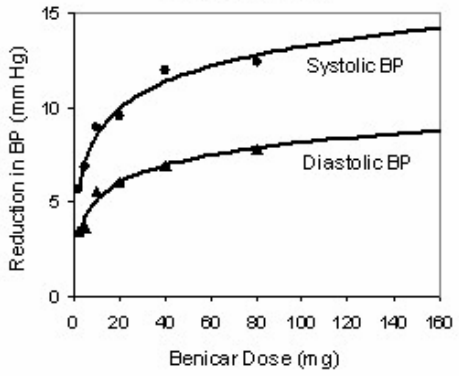
The blockade of A-II Type 1 receptors has been shown (in-vitro) to reduce production of the Th1 cytokines, including TNF- α , an action which would interdict this inflammatory process.

Angiotensin Receptor Blockers are currently indicated for hypertension. There is thus a simple clinical test of this hypothesis available for patients who are suffering both from Sarcoidosis and mild hypertension.

We have found that Benicar (Olmesartan Medoxomil), administered as 40mg every 6 to 8 hours, provides a very effective angiotensin blockade.

There are two issues in the selection and dosage of the ARB. As you can see from the pressor effect vs. dosage for Benicar (Figure 1), about 90% of the ultimate pressor effect can be achieved with only 40mg, once per day. But this dose is not well tolerated by sarcoid patients. Partly this may be the result of the additional Angiotensin receptors in the inflamed tissues, all of which have to be blocked, and partly it may be due to an increased production of serum

Figure 3. Placebo-Adjusted Reduction in Blood Pressure



ACE by macrophages in response to the partial blockade. Sarcoid patients experience symptoms ranging from increased fatigue to psychedelic dreams when prescribed ARBs just once daily, the customary prescription for hypertension

To be fully effective, we found that Benicar must be prescribed to sarcoidosis patients as 40mg every 6 to 8 hours. We found the ARB Diovan (Valsartan) to be less effective than Benicar, but it may be used at the 80mg q8h described in our earlier paper. Two patients reported sinus congestion with the Diovan blockade, which was not present after changing to Benicar. Controlled studies are needed to accurately define the blockade capability of each ARB, individually and in combination.

In summary, Dr. Marshall's discovery reveals that during the pathogenesis of borreliosis the BLPs of borrelia trigger inflammation via TLR signaling pathways or by other intracellular activation of NF-kappa B, which stimulates the gene expression for inflammatory cytokines. When the inflammatory cascade goes chronic, it eventually goes into the self-perpetuating cycle described by Marshall. This cycle will continue to produce disease until it is stopped by

intervention.

In my opinion, Dr Marshall's discovery that A-II perpetuates a TH1 inflammatory cascade is nothing short of major medical breakthrough.

Dr. Marshall's work has not only given us the model for the pathogenesis, he has also given us the therapeutic approach that breaks the perpetual cycle that maintains the inflammatory cascade.

Angiotensin II type 1 receptor blockade. The angiotensin receptor blocker (ARB) called Benicar (olmesartan medoxomil) has specific ARB properties that block this self-perpetuating inflammatory cascade.

Benicar therapy is a medical miracle for those suffering with chronic borreliosis.

Inducible Nitric Oxide synthase (iNOS) and its role in pathogenesis

TLR activation of the a transcription factor known as NF-kappa B stimulates the gene expression of inducible nitric oxide synthase (iNOS). In this way, BLPs can trigger an increased production of nitric oxide as the following reference note:

Vet Immunol Immunopathol. 1999 Feb 22;67(3):271-84.

Up-regulation of inducible nitric oxide synthase mRNA in dogs experimentally infected with Borrelia burgdorferi.

Harter L, Straubinger RK, Summers BA, Erb HN, Appel MJ.

James A. Baker Institute for Animal Health, College of Veterinary Medicine, Cornell University, Ithaca, NY 14853, USA.

Comparing the number of borrelia-positive tissues in dogs within Group A (acute lameness) it was interesting to note that the dogs investigated on the first day of lameness showed more borrelia-positive tissues than dogs from Days 2±4 of lameness. The mean of the seven dogs with 1-day lameness was 72%, whereas the mean of the 3-day lameness dogs was 36% (the single value for the 2-day lameness is 48%, for the 4-day lameness is 36%). This apparent decrease of the borrelia load during an episode of acute Lyme arthritis may be a result of the host's inflammatory response. In contrast, the number of iNOS-positive tissues increased from 40% for dogs with 1-day lameness to 64.3% for the dog A96-2/3 with the 4-day lameness. While this might suggest the role for NO in clearing B. burgdorferi from the infected tissues, the number of dogs investigated was not sufficient to establish a reliable relationship between the borrelia load and the number of iNOS-positive tissues during lameness. Further studies should clarify this point. The milder and generally subclinical chronic Lyme arthritis, as represented by dogs from Group B, is characterized by a lower number of borrelia-positive tissues and a lower number of iNOS-positive tissues as well.

Immunol Lett. 1994 May;40(2):139-46.

Killing of Borrelia burgdorferi by macrophages is dependent on oxygen radicals and nitric oxide and can be enhanced by antibodies to outer surface proteins of the spirochete.

Modolell M, Schaible UE, Rittig M, Simon MM.

Max-Planck-Institut fur Immunbiologie, Freiburg, Germany.

Interaction of B. burgdorferi organisms with mouse bone marrow-derived macrophages (BMM phi) leads to phagocytosis of microorganisms, induction of nitric oxide (NO) and superoxide radicals (O2-) by BMM phi and killing of spirochetes. Destruction of spirochetes by BMM phi was quantified by a new method based on the release of radioactivity from spirochetes pre-labelled with [3H]adenine. Uptake of B. burgdorferi by BMM phi, which mainly occurs by coiling phagocytosis, generation of NO and O2- radicals as well as killing of spirochetes were significantly enhanced by pre-opsonization of spirochetes with monoclonal antibodies (mAb) to the outer surface proteins A and B but not with those to the periplasmic flagellin. Addition of inhibitors specific for NO and O2- radical synthesis either separately or together to cultures of BMM phi and spirochetes resulted in only partial reduction of the killing potential of effector cells. The data indicate that NO and O2- radicals are necessary, but not sufficient, for complete elimination of B. burgdorferi by macrophages. Together with previous findings that protection against B. burgdorferi infection is conveyed by humoral immune responses the present data indicate that one of the important functions of specific antibodies is their participation in macrophage-mediated control of spirochetes.

Decreased nitric oxide-mediated natural killer cell activation in chronic fatigue syndrome.

Ogawa M, Nishiura T, Yoshimura M, Horikawa Y, Yoshida H, Okajima Y, Matsumura I, Ishikawa J, Nakao H, Tomiyama Y, Kanayama Y, Kanakura Y, Matsuzawa Y.

Osaka University Medical School, Osaka, Japan.

BACKGROUND: L-Arginine (L-Arg), one of the essential amino acids, has been reported to have an immunomodulatory effect. The precise mechanism of the L-Arg-induced natural killer (NK) cell activation remains unresolved,and the effect of L-Arg on NK cells in chronic fatigue syndrome (CFS) patients has not been estimated. METHODS: NK cell function was evaluated in 20 subjects with CFS and compared with that in 21 healthy individuals. RESULTS: In healthy control subjects, NK activity was significantly increased after treatment with L-Arg, an NK function enhancer, for 24 h, whereas the same treatment failed to enhance NK activity in the CFS patients. We thus focused on L-Arg metabolism, which involves nitric oxide (NO) production through NO synthase (NOS). The expression of inducible NO synthase (iNOS) transcripts in peripheral blood mononuclear cells was not significantly different between healthy control subjects and CFS patients. The L-Arg-mediated NK cell activation was abolished by addition of NG-monomethyl-L-arginine, an inhibitor for iNOS. Furthermore, incubation with S-nitroso-N-acetyl-penicillamine, an NO donor, stimulated NK activity in healthy control subjects but not in CFS patients. CONCLUSION: These results demonstrate that the L-Arg-induced activation of NK activity is mediated by NO and that a possible dysfunction exists in the NO-mediated NK cell activation in CFS patients.

Infect Immun. 1994 Sep;62(9):3663-71.

Outer surface lipoproteins of Borrelia burgdorferi stimulate nitric oxide production by the cytokine-inducible pathway.

Ma Y, Seiler KP, Tai KF, Yang L, Woods M, Weis JJ.

Department of Pathology, University of Utah School of Medicine, Salt Lake City 84132.

The outer surface lipoproteins of Borrelia burgdorferi, OspA and OspB, stimulate the production of nitric oxide (NO) by murine bone marrow-derived macrophages from BALB/c, C3H/HeN, and C3H/HeJ mice. Gamma interferon (IFN-gamma) caused a three- to fivefold enhancement of this production of NO, and the L-arginine analog N-guanidino-monomethyl L-arginine inhibited it. Activation of transcription of the inducible NO synthase gene in stimulated macrophages was demonstrated by reverse transcriptase rapid PCR. Although IFN-gamma increased the amount of NO produced in macrophage cultures, it did not cause transcription of the inducible NO synthase gene greater than that seen with the Borrelia proteins. OspA and OspB also induced the production of high levels (40 to 150 ng/ml) of IFN-gamma in cultures of macrophages incubated with interleukin-2 (IL-2)-elicited cells from normal (T and NK cells) and scid (NK cells) mice but not in macrophages or IL-2-elicited cells cultured individually.
This suggests that OspA stimulated macrophage production of cytokines, which, in turn, stimulated the production of IFN-gamma by NK and T cells. Reverse transcriptase rapid PCR demonstrated that OspA and sonicated B.

burgdorferi stimulated production of several inflammatory cytokines in macrophage cultures, including IL-1, IL-6, IL-12, IFN-beta, and tumor necrosis factor alpha. As tumor necrosis factor alpha, IFN-beta, and IL-12 are potent activators of IFN-gamma production by T and NK cells, their presence in these cocultures could be responsible for the IFN-gamma production. Lymphocytes from infected C3H mice also produced IFN-gamma when stimulated with B. burgdorferi; thus, immune cells may also modulate NO responses. The generation of NO during infection with B. burgdorferi may be important, as NO has potent antimicrobial properties. NO can also be involved in pathological inflammatory processes in which its generation is detrimental to the host. Thus, the colocalization of B. burgdorferi lipoproteins, NO-producing cells, and regulatory cytokines may determine the outcome of infection.

Borrelia lack the microbial toxins called lipopolysaccharides (LPS) however, they have over 150 genes that encode for the BLPs that are the key to their pathogenicity. This is over 50 times greater than other pathogenic bacteria. That is, other bacteria usually only have 3 genes for lipoproteins, while borrelia have 150!

With this many BLPs triggering an imbalance of the immune system and other innate responses in the body, it's not hard to see how a cascade of chronic problems can arise from this.

For example, when we look at psychological problems of neuroborreliosis, it's clear that the cause of these symptoms arise from BLPs triggering encephalitis and that this inflammation not only causes imbalances in numerous neurotransmitters, it also causes vasculitis which leads to hypoperfusion and hypoxia of the brain.

These toxins also cause a channelopathy, which lead to a dysfunction of signals along neurons, muscles and cells making them easily excitable, but not able to discharge correctly…since the electrical potential across the cell membranes don't function normally.

This can attribute to numerous symptoms such as anxiety, paresthesias, hyperacusis, tremors and even susceptibility to static shock that many LD patients have.

Thus, the evidence is pretty clear that BLPs plays a large role in the pathogenesis of borreliosis and is a key to understanding this disease.

IMO, without BLPs, borrelia would not be virulent.

Some questions I've received regarding the pathogenesis of borreliosis:

Q: I don't see anything so far that would describe why these inflammatory mediators, which would be activating the T-cells, would not be phagocytized by these and other macrophages.

A: Initially it does. The initial infection of borrelia can be acute and flu-like or it may not even be noticable. Unfortunately, this initial immune response doesn't completely rid the body of borrelia.

I don't think we have the complete answer to why this happens. But there are probably several factors involved. Borrelia can adapt and be very stealthy. Another reasonable theory is that apoptosis of monocytes, macrophages, neutrophils and other immune cells triggered by TLR-2 stimulation may account for immune dysfunction and supression.

This makes a lot of sense to me since we see immune dysfunction & suppression in chronic LD and in CFIDS, which I believe have a very similar pathogenesis.

Q: Also, if it's somehow a more severe activation of these mediators, especially if caspase and IL-12 are involved, why is there not more obvious toxic shock-like symptoms apparent early on?

A: I wouldn't say that it's a more severe activation of these mediators, but more of a long term chronic/relapsing imbalance of them.

It may also have to do with borrelia's ability to grow slowly and not trigger an acute toxic shock.

It also may have something to do with borrelia's ability to hold on to it's BLPs and not release them into the surrounding tissue until they are killed.

We know that when we kill borrelia (such as with abx tx) the BLPs are released from them and we experience a herx reaction. In severe cases this response can be quite acute.

There are probably several other factors such as lipid depot effect and slow release.

The immune supressing effect of BLPs likely plays a role here too.

Q: It seems that many people are not even symptomatic until years after the original tick exposure.

A: This is true with borreliosis...(this happened in my case). It's also true with syphilis.

This may be due to borrelia's ability to code for so many different BLPs. They have the genetic code for over 150 different BLPs.

Depending on which ones and how strong the expression of these BLPs genes are may determine how virulent the borrelia is at different times.

We know that borrelia change the expression of these genes when exposed to different environmental factors such as temperature.

They express different BLPs in ticks since they are in ambient temperature, but once inside a mammal, they begin to change the BLPs that they express.

They also have an effective ability to accept plasmids and pick up other pathogenic genes in this way.

Work has shown that removing certain BLPs from virulent strains makes the borrelia avirulent.

The evidence is very suggestive that BLPs determines the pathogenesis of borrelia.

This work has appeared very timely for this discussion:

J Infect Dis. 2004 Jan 1; 189(1): 113-9. Epub 2003 Dec 22

Selective Induction of Matrix Metalloproteinases by Borrelia burgdorferi via Toll-Like Receptor 2 in Monocytes.

Gebbia JA, Coleman JL, Benach JL.

Center for Infectious Diseases, Centers for Molecular Medicine, and State of New York Department of Health, Stony Brook University, Stony Brook, New York, USA.

Regulation of secretion of matrix metalloproteinase (MMP) underlies the basis of numerous physiological and pathological processes in multicellular organisms.

The Toll receptor family, which is conserved from Drosophila species to humans, mediates pattern recognition of a diversity of ligands involved in morphogenesis and innate immunity. Here, we show that secretion of MMP-9 is selectively induced through Toll-like receptor (TLR) 2 in human and murine monocytic cells stimulated with Borrelia burgdorferi.

Secretion of MMP-1 was shown to be stimulated through a pathway other than TLR2, under identical conditions. Analysis of nuclear extracts indicated that activator protein (AP)-1 was reduced in TLR2-neutralized monocytic cells, suggesting that AP-1 plays a role in the transcriptional activation of MMP-9 through TLR2.

The specific induction of MMP-9 through TLR2 provides direct evidence of a new role for this ancient receptor family in regulating secretion of MMPs and demonstrates evolutionary convergence between invertebrate morphogenesis and the vertebrate innate immune system.

Gulf-War-Syndrome (GWS) has very similar symptoms to chronic borreliosis.

Mycoplasma fermentans is being considered to be the likely pathogen...whether it is or not, it's likely some BLP producing microbe is.

Since the symptoms are similar, I'd expect the pathogenesis would be similar.

Work such as this indicates that this may be the case:

Stimulation of human Toll-like receptor (TLR) 2 and TLR6 with membrane lipoproteins of Mycoplasma fermentans

induces apoptotic cell death after NF-kappaB activation.

Into T, Kiura K, Yasuda M, Kataoka H, Inoue N, Hasebe A, Takeda K, Akira S, Shibata KI.

Departments of Oral Pathobiological Science and Oral Health Science, Hokkaido University Graduate School of Dental Medicine, Nishi 7, Kita 13, Kita-ku, Sapporo 060-8586, Japan. Department of Host Defense, Research Institute for Microbial Diseases, Osaka University, 3-1 Yamada-oka, Suita, Osaka 565-0871, Japan.

Mycoplasmal membrane diacylated lipoproteins not only initiate proinflammatory responses through Toll-like receptor (TLR) 2 and TLR6 via the activation of the transcriptional factor NF-kappaB, but also initiate apoptotic responses.

The aim of this study was to clarify the apoptotic machineries. Mycoplasma fermentans lipoproteins and a synthetic lipopeptide, MALP-2, showed cytocidal activity towards HEK293 cells transfected with a TLR2-encoding plasmid.

The activity was synergically augmented by co-expression of TLR6, but not by co-expression of other TLRs. Under the condition of co-expression of TLR2 and TLR6, the lipoproteins could induce maximum NF-kappaB activation and apoptotic cell death in the cells 6 h and 24 h after stimulation respectively.

Dominant-negative forms of MyD88 and FADD, but not IRAK-4, reduced the cytocidal activity of the lipoproteins. In addition, both dominant-negative forms also downregulated the activation of both NF-kappaB and caspase-8 in the cells.

Additionally, the cytocidal activity was sufficiently attenuated by a selective inhibitor of p38 MAPK.

These findings suggest that mycoplasmal lipoproteins can trigger TLR2- and TLR6-mediated sequential bifurcate responses: NF-kappaB activation as an early event, which is partially mediated by MyD88 and FADD; and apoptosis as a later event, which is regulated by p38 MAPK as well as by MyD88 and FADD.

In regards to this and other work showing that BLPs stimulates MMP-9, what this means is that the BLPs of borrelia have the ability to turn our own immunes system against the extracellular proteins of our body.

Extracellular proteins (ECP) make up the "mortar" between our cells, ie, it's what holds the cells together in tissues.

There are several proteins in ECP: collagen, elastin, gelatin, and several others.

BLPs have the ability (thru TLR-2) to trigger our immune system to produce and release protease enzymes that digest these important ECPs. These include proteases such as collegenase, and elastase.

This protease activity is very damaging to tissues and itself can stimulate even more inflammation.

Borrelia takes advantage of these protease enzymes to penetrate through the tissues of the body. As ECPs are digested in tissues it is easier for borrelia to move through it.

This portion of the pathogenesis of borrelia explains a lot of the fibromyalgia symptoms in LD.

IMO, FMS/CFIDS and borreliosis/Lyme disease have a similar pathogenesis, which is TLR triggering by the BLPs or LPS of pathogenic microbes.

Borrelia also have an affinity for the endothelial cells lining the inside of blood vessels. They attach and infect the endothelial cells of blood vessels and trigger inflammation in these infected blood vessels (vasculitis) wherever they reside in the body. In other words, once the Borrelia infects the endothelial cells, they stimulate vasculitis of the blood vessel. Vasculitis of the nervous system, skin, muscle, tendons, and connective tissue explains much of the disease and clinical symptoms caused by Borrelia.

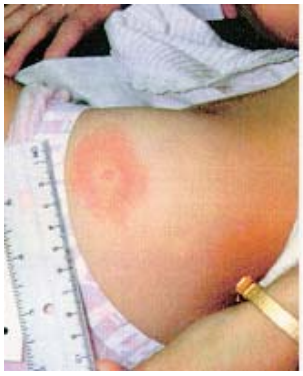
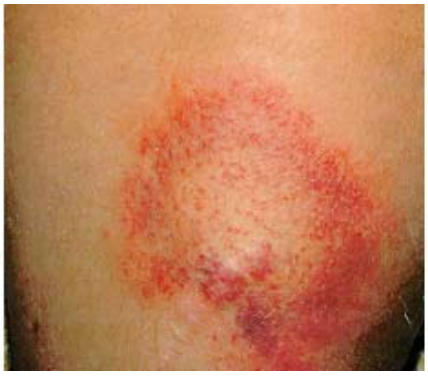
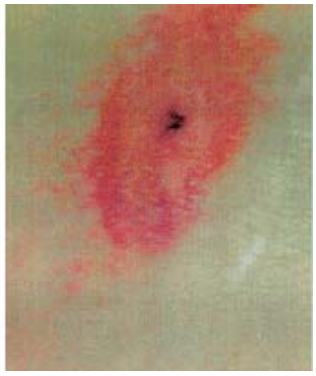
Borrelia species have several other characteristics that make them a challenging pathogen. They can reside and grow intracellularly (inside the cells); out of reach of the host's immune system. By rotating their filaments, the spirochetes are able to move throughout the body in a corkscrew fashion; invading tissue, penetrating cells, replicating, and destroying the host cells they infect. The spirochetes hide their flagella, which are normally antigenic, from the host immune defenses. As Borrelia emerge from host cells they can wrap themselves with the membrane of the host cell, thus concealing themselves from the hosts immune system.

Borrelia's ability to grow slowly and avoid attack by the host's immune system makes it a difficult pathogen to

destroy. Not only can borrelia evade the host immune system, but often will suppress it as well.

It's also important to remember that borellia can go dormant in the body for lengthy periods of time. Then it can return to cause disease at any time.

The Early Symptoms of Lyme Disease

It's important to remember that *fewer than 50%* of patients with LD will initially have the skin rash called erythema migrans (EM). An EM rash with a round red ring and a central clearing (see figure 10) is called a bulls-eye rash and it is diagnostic for Lyme disease. In other words, a bulls-eye rash is enough evidence to confirm a diagnosis of LD. However, I emphasize that EM is seen in less than 50% of infected individuals with Lyme disease. The reason I stress this is because many physicians think they must see an EM rash and have laboratory confirmation before they will make a diagnosis of Lyme disease. But, if less than 50% of LD cases have an EM rash, physicians misdiagnose these early clinical cases of Lyme disease and the prognosis for these mistreated patients deteriorates with time.

Figure 10: The presentation of erythema migrans in early borreliosis

If present, the EM rash appears between 3 days to 1 month following the bite of an infected tick; however, the rash typically resolves itself spontaneously over a 2-4 week period. The EM rash grows concentrically over the following 5-10 days and without treatment may last for up to several weeks. The rash can vary from very small to very large (up to twelve inches across). Unfortunately, the EM rash is not the only rash associated with Lyme. Various other rashes associated with LD have been reported. One tick bite can cause multiple rashes. The rash can mimic such skin problems as hives, eczema, sunburn, poison ivy, fleabites, etc. The rash can itch, feel hot or it may even be asymptomatic and go unnoticed. The rash can disappear and return several weeks later. I recommend taking a photograph of any rash, especially if associated with a tick bite. The photos will help the Lyme knowledgeable physician make a proper diagnosis and prescribe the appropriate treatment.

All rashes that occur at the site of a tick bite are not due to Lyme disease. An allergic reaction to tick saliva often occurs at the site of a tick bite. This rash can be confused with the rash of Lyme disease. Allergic reactions to tick saliva usually occur within hours to a few days after the tick bite; they usually do not expand and disappear within a few days. EM rashes caused by Lyme disease persist longer, but usually subside within a few weeks.

The occurrence of multiple EM skin rashes is indicative of systemic spread of the organisms. Multiple EM lesions usually do not occur until after 2-4 weeks following the initial tick bite. This is the same time period during which the organisms are being spread throughout the body to other tissues and cells.

Borrelia Slowly Spreads Throughout the Body --
Approximately 4-6 weeks following the tick bite, the first systemic signs of Lyme disease that may or may not occur are in the form of flu-like symptoms or malaise. These symptoms include sore throat, severe headaches and neck aches, severe fatigue, chills and fever, and swollen lymph nodes. Upper respiratory symptoms are usually not present with LD, distinguishing it from other flu-like illnesses. While the LD-flu symptoms can spontaneously resolve themselves, patients can experience relapses.

Soon after the onset of Lyme-flu, arthralgias and/or myalgias (muscle and joint pain) may begin. The arthralgias appear to primarily involve the large joints (i.e. knees, elbows, hips, shoulders), although smaller joints (e.g. wrists, hands, fingers, toes) may be involved. The pains are generally described as severe, jumping from joint to joint, and may be present for only short periods of time. Pain in the teeth and in the temporal-mandibular joints (jaw) is common. Neurological involvement will cause associated paresthesias (muscle twitching, burning sensations, prickling / shooting pains, and numbness). Lyme disease can cause palsy of affected areas, but it's more likely to cause neurosensory deficits before neuromotor disease.

Facial nerve (Bell's) palsy is another neurological symptom of Lyme disease. Encephalitis or encephalopathy may manifest as cognitive dysfunction, including short-term memory loss, and psychiatric symptoms such as panic, anxiety, or depression. The encephalitis and facial paralysis tend to occur within the first few months following the tick bite, but may also occur as part of a relapse at any time.

Other symptoms in this stage of the disease may include blurred vision, uveitis, ringing in the ear (tinnitus, which was one of my first symtoms) and/or hearing loss, shortness of breath, palpitations or tachycardia (rapid heart rate), chest pains, abdominal pains, diarrhea or irritable bowel, testicular or pelvic pain, urinary incontinence/urgency, dizziness, tremors, dysautonomia, and hepatitis.

Borreliosis: The Clinical Disease

Lyme disease is an extremely challenging infectious/toxic disease for both doctor and patient. It can exhibit many different symptoms. The clinical picture of LD can be similar to fibromyalgia, including: chronic fatigue, joint pain (arthralgias), muscle, fibrous tissue and tendon pain. Lyme disease can also manifest primarily as a neurological disorder, including fatigue and many neurological symptoms. It is important to remember that there are hundreds of symptoms that are caused by LD and it can mimic many diseases; for this reason, LD is often called, " the great imitator."

The prognosis of Lyme disease depends a lot on how soon the disease is caught and how well it is treated. Early, aggressive, and comprehensive treatment improves the prognosis tremendously. Unfortunately it is difficult to diagnose many cases early because they don't present themselves with obvious Lyme disease symptoms. They often show only one or a few subtle symptoms that can easily be misdiagnosed as something else.

Lingering Lyme: The Chronic Persistent Infection

Some symptoms and signs of Lyme disease may not appear until weeks, months, or years after a tick bite. This stage typically involves intermittent episodes of joint pain or numerous neurological symptoms such as: meningitis, Bell's palsy, dysfunction of cardiac rhythm, and migratory pain to joints, tendons, muscle and bone. Arthritis is most likely to appear as brief bouts of pain and swelling, usually in one or more large joints, especially the knees. In some patients, the first and only sign of Lyme disease is arthritis. In others, nervous system problems are the only evidence of Lyme disease. However, any combination of symptoms can be present.

Primarily unique to humans, neuroborreliosis (the neurological form of Lyme disease) can include numbness, pain, Bell's palsy (paralysis of the facial muscles, usually on one side and more often the left), and meningitis (fever, stiff neck, and severe headache).
Dysautonomia (a dysfunctioning autonomic nervous system) and irregularities of the heart rhythm may occur.

In a minority of individuals (11%), development of chronic Lyme arthritis may lead to erosion of cartilage and/or bone. Other clinical manifestations associated with chronic neuroborreliosis include neurologic complications such as disturbances in memory, mood, or sleep patterns, and sensations of numbness and tingling in the hands or feet (parethesia).

The course of the disease can best be described as persistent, with periods of worsening symptoms, often cyclical every few weeks or monthly. Especially disconcerting are persistent symptoms such as pain, headaches and fatigue. Some patients are more symptomatic than are others, which may reflect gender and genetically-determined differences in response to infection. The disease is progressive, destructive, and debilitating, and in severe untreated cases, it can be fatal.

Chronic Borrelia can also cause a degenerative skin disorder now known as acrodermatitis chronica atrophicans (ACA).

Lyme disease causes metabolic/endocrine dysfunctions that lead to weight loss or commonly chronic weight gain.

Generally, women struggle with chronic Lyme disease more severely than men do. It is not known for sure why.

List of Lyme Disease Symptoms

As I mentioned before, every organ and organ system can be affected, here's a list of some of the LD symptoms as they relate to specific areas of the body:

- Head – headache, neck pain, facial pain and paralysis, difficulty chewing, pain in teeth, dry mouth, loss of

taste/smell, numb tongue/mouth. Peculiar metallic or salty taste is also common in LD. This is likely due to the BLPs present in the system.

- Bladder -- frequent or painful urination, repeated urinary tract infections, irritable bladder, interstitial cystitis.
- Lung -- respiratory infection, cough, asthma, pneumonia, pleurisy, chest pains
- Ear -- pain, hearing loss, ringing (tinnitius), sensitivity to noise, dizziness & equilibrium disorders.
- Eyes -- pain due to inflammation (scleritis, uveitis, optic neuritis), dry eyes, sensitivity to light, drooping of eyelid (ptosis), conjunctivitis, blurry or double vision, swelling around eyes / bags below the eyes.
- Throat -- sore throat, swollen glands, cough, hoarseness, difficulty swallowing
- Neurological -- headaches, facial paralysis, seizures, meningitis, stiff neck, burning, tingling, or prickling sensations (parathesia), loss of reflexes, loss of coordination, equilibrium problems/dizziness (these symptoms mimic an MS, ALS, or Parkinson' s like syndrome)
- Stomach -- pain, diarrhea, nausea, vomiting, abdominal cramps, anorexia
- Heart -- weakness, dizziness, irregular heart-beat, myocarditis, pericarditis, palpitations, heart block, enlarged heart, fainting, shortness of breath, chest pain, mitral valve prolapse.
- Muscle & skeletal system -- arthralgias (joint pain), fibromyalgia (muscle inflammation and pain)
- Other Organs -- liver infection / hepatitis, elevated liver enzymes, enlarged spleen, swollen testicles, and irregular or ceased menses.
- Neuropsychiatric -- mood swings, irritability, anxiety, rage (Lyme rage), poor concentration, cognitive loss, memory loss, loss of appetite, mental deterioration, depression, disorientation, insomnia

- Pregnancy -- miscarriage, premature birth, birth defects, stillbirth
- Skin – EM, single or multiple rash, hives, ACA
- Another interesting symptom often noticed is an increased susceptibility to electrostatic shock. This is likely due to the BLPs causing a change in the electro-potential in our cells/nervous system. Some of these toxins are likely sodium channel agonists and can change the electrical potential of our body. Thus, the likelihood of electro-static shock.

One or more of these symptoms is not diagnostic for LD, except for a bulls-eye EM rash. A diagnosis for LD is a clinical one and must be made by a physician experienced in recognizing LD symptoms and history, experienced in interpreting lab results and recognizing a response to treatment. Always remember that negative serological tests are not reliable and cannot be used solely for a diagnosis. These tests frequently are incorrectly negative.

The Diagnosis of Lyme Disease

Lyme disease is diagnosed clinically based on history, clinical symptoms, and response to therapy. No test can conclusively "rule-out" Lyme disease. It is critical to understand that the diagnosis of Lyme disease is heavily weighed on clinical symptoms and history alone. LLMDs are familiar with the complex nature of Lyme disease and are very aware of the subtle symptoms it can produce. Their clinical judgment must be very keen for them to recognize early, subtle cases.

Clinically, "chronic fatigue syndrome" or "fibromyalgia", which is more recently called "chronic fatigue immune dysfunction syndrome" (CFIDS) cannot be readily distinguished from chronic Lyme disease and in fact is probably one in the same disease. Yes, I believe along with many LLMDs that most cases of fibromyaliga and CFIDS are actually misdiagnosed cases of chronic Lyme disease. Strong support for this comes from the fact that antibiotic and other LD therapies improve many patients diagnosed with fibromyalgia.

Routine laboratory tests are usually normal in LD. Liver enzymes may be elevated from hepatitis. The erythrocyte sediment rate (ESR) is most often normal, distinguishing it from some of the purely inflammatory disorders such as rheumatoid arthritis or lupus. However, overlap between LD and autoimmune diseases frequently occur. I believe that the chronic inflammation and immune dysfunction caused by LD often leads to autoimmune diseases. Culture of the Borrelia is rarely possible but can occur in a few early LD cases of B. burgdorferi, usually from biopsies of the EM rash. However, most laboratories are not capable of the difficult culturing of these slow growing borrelia organisms and we have not been able to isolate many borrelia species in the laboratory.

Currently available serological (blood) tests for LD caused by B. burgdorferi include the immunologically-based ELISA and Western blot assays. The problem with the ELISA is the high amount of background compared to Western blot assays, likely due to the use of whole organisms. After correction for the high background, only a small percentage of positives can be detected. Because the Western blots separate the proteins of the borrelia, specific reactions can be seen, and more accurate interpretations of the results made. Clinically over 75% of patients with Lyme disease are negative by ELISA, but positive by Western blot. However, it is important to remember that there are many patients (approximately 30%) who have symptoms, but whose Western blots are negative. The

different antigenic profile between Borrelia species along with their ability to avoid the immune system and remain intracellular could explain the absence of immune responses. Even more likely is that the pathology of borreliosis can lead to immune dysfunction. Cells of the immune system can be triggered into apoptosis (preprogrammed death or suicide). The death of cells of the immune system can also explain the dysfunctional immune system and the problems there are using serological laboratory tests to diagnose chronic borreliosis as shown below:

Epidemiol Mikrobiol Imunol. 2001 Feb;50(1):10-6.

Persistence of Borrelia burgdorferi sensu lato in patients with Lyme borreliosis

Honegr K, Hulinska D, Dostal V, Gebousky P, Hankova E, Horacek J, Vyslouzil L, Havlasova J.

Infekcni klinika, Fakultni nemocnice, Hradec Kralove.

In 18 patients with Lyme borreliosis the authors proved the persistence of Borrelia burgdorferi sensu lato by detection of the causal agent by immune electron microscopy or of its DNA by PCR in plasma or cerebrospinal fluid after an interval of 4-68 months. Clinical manifestations common in Lyme borreliosis were present in only half the patients, in the remainder non-specific symptoms were found. In nine subjects with confirmed Borrelia burgdorferi sensu lato in the cerebrospinal fluid the cytological and biochemical finding was normal. ***Examination of antibodies by the ELISA method was negative in 7 of 18 patients during the first examination and in 12 of 18 during the second examination.***
In all negative examinations the specific antibodies were assessed by the Western blot or ELISA method after liberation from the immunocomplexes.
In the authors' opinion it is advisable to examine repeatedly plasma and other biological material from potentially affected organs by PCR and subjects with persisting or relapsing complaints after the acute form of Lyme borreliosis as well as to examine cerebrospinal fluid in case on non-specific symptoms and concurrent pathic EEG or MR findings.

There is a critical need for the development of a better diagnostic test to detect Lyme disease caused by Borrelia species. Until then, the Western blot assay is the best clinical test currently available.

PCR (Polymerase Chain Reaction) is a highly sensitive way to detect microbial DNA and is beginning to play an important role in the diagnosis and research of borreliosis. This technology has the potential to bridge the gap between the clinical diagnosis and the laboratory confirmation.

The Treatment of Lyme Disease

Our understanding of treating this disease has made a great leap recently with the discovery of Dr. Marshall's work.

The best approach to therapy is a multifaceted and comprehensive treatment regimen that includes conventional antibiotics, numerous complimentary and alternative therapies, and a nutritional regimen specifically designed to reduce inflammation, aid the liver in detoxifying the BLPs, decrease the risk of yeast infections, repair damage caused by the disease and strengthen the immune system.

The mainstay of treating chronic borreliosis is to follow Dr. Marshall's protocol for sarcoidosis found here: www.sarcinfo.com
. This comprehensive therapeutic approach is much more effective than the conventional treatment that uses only a single therapeutic entity such as antibiotics alone.

Antimicrobial Therapy: **The Conventional Treatment**
-- Antibiotics are the foundation of conventional Lyme disease therapy. We are often told by the medical establishment and the general media that Lyme disease is easily treated with antibiotics. Unfortunately, in most cases, this is not true. If caught early, then yes, Lyme disease can be treated relatively well with only antibiotics. But, as I mentioned earlier, most of the time it's not caught early and once the borrelia has spread throughout the body and secondary symptoms have occurred, treatment gets much more complicated and less effective.

Oral therapy with doxycycline, minocycline, tetracycline or amoxicillin is appropriate for early cases of Lyme disease. Parenteral therapy, usually intravenous (IV) administration, maybe used for patients with neurologic involvement, severe arthritis, or any life-threatening manifestation of Lyme disease such as complete heart block. However, there's good evidence to suggest that oral therapy can be just as effective if not more so in most cases.

If you can't find a LLMD in your area and can't get in to see one on a timely basis there are places you can order

prescription medication without a prescription such as this: http://www.edrugnet.com or www .rxsolutions.com .

The treatment of patients with chronic Lyme disease is very controversial. The more I study the chronic symptoms of Lyme disease, the more I believe that the persistence of the disease is caused by a persistent borrelia infection and the persistent or residual presence of fat-soluble BLPs that can depot in the lipid tissues throughout the body and in many cases lead to immune dysfunction.
Permanent damage can also occur throughout different stages of disease and cause chronic symptoms to persist.

Borrelia species are sensitive to several oral antibiotics including: amoxicillin, tetracycline, doxycycline, minocycline, clarithromycin (Biaxin®), metronidazole (Flagyl®), co-trimoxazole sulfamethoxazole/trimethoprim (Bactrim® or Septra®) and azithromycin (Zithromycin®).

Early in Lyme disease at the time of the early skin EM rash, any one of the above antibiotics appears to be effective if given for at least 2 months. Antibiotic treatment of only 2-3 weeks is insufficient and patients often deteriorate with chronic symptoms of borreliosis including; arthralgias, fatigue, and paresthesias. Thus, LLMDs recommend that tetracycline, doxycycline, minocycline or amoxicillin be used for at least 2 months if an EM rash is the first and only symptom of Lyme disease.

If any other symptoms appear after an EM rash, the treatment of LD for only 2-4 weeks frequently fails and relapses occur. LLMDs suggest that several months of doxycycline, minocycline or other appropriate antibiotics have a higher success. In patients with symptoms present for more than six months, the treatment course may need to be more prolonged, or a retreatment course of varying length may be needed. In patients with symptoms for more than a year, several months or years may be needed for complete resolution of symptoms. In general, treatment for chronic LD usually requires a period of time equal to or greater than the time the patient has had the disease. The slow growth rate and the cryptic and stealthy nature of borrelia justify the need for longer antimicrobial treatment periods.

When Some Symptoms are a Diagnostic Sign: The Jarisch-Herxheimer Reaction to Treatment

Once treatment is initiated for patients beyond the EM stage, their symptoms frequently increase during the first several days, or even for the first two weeks of therapy. This reaction is called a Jarisch-Herxheimer reaction (herx) and is the body's response to the BLPs being released from the dying borrelia. These BLPs stimulate the inflammatory cascade which explains this phenomenon. The herx reaction is a very helpful clinical symptom Lyme clinicians use to support a clinical diagnosis of Lyme disease.

For patients with preexisting symptoms of more than a few months, relief of any of their symptoms may not occur until after 4-6 weeks of therapy. Typically, there are short periods of relief, followed by relapsing or migrating symptoms. With continued therapy, there are longer symptom-free periods. Some arthralgias may require several months of therapy to resolve, and fatigue and dizziness may be the last symptom to resolve.

The preference for a variety of antibioitics by some LLMDs is based on a large number of failures that were noted in patients who had been on only amoxicillin, or doxycycline. It seemed that patients generally have some initial relief, but then plateau. It's not clear if tetracycline is as effective as doxycycline; some claim it is not while others say it is better. There are some physicians who use doxycycline at high doses of 600mg daily to achieve a more successful result. It's my opinion that low to moderate doses of antibiotics is more appropriate. See the work of Dr. Brown: www.roadback.org .

Metronidazole (Flagyl) is a very effective antimicrobial for treating chronic Lyme disease. It distributes well throughout the body and is able to penetrate tissue and cells. This ability allows metronidazole to reach the cryptic borrelia throughout the body and kill it. Metronidazole is also effective at attacking the cyst form of borrelia. This may be the single most effective antimicrobial pharmaceutical for treating Lyme disease. The disadvantage of metronidazole is it's toxicity to the liver and neurological system. It can raise liver enzymes and cause peripheral neuropathies similar to LD itself. These side-effects must be prudently monitored.

Of the beta lactams used for the treatment of Lyme disease, the most effective appears to be ceftriaxone (Rocephin). In limited comparitive trials, cefotaxime appears to be equally effective, and high-dose IV penicillin is too. In early stages of LD, oral amoxicillin is as effective as doxycycline. In later stages, however, many failures are noted, despite the use of up to 3 grams of amoxicillin daily.

The newer macrolides such as azithromycin, roxithromyin and clarithromycin are effective. LLMDs often use clarithromycin and azithromycin in higher dosages for longer periods of time than regularly recommended for these antibiotics.

In evaluating all of the possible factors, it would appear that antibiotics that can inhibit the bacteria's ability to synthesize toxic bacterial lipoproteins and achieve intracellular concentrations are the most efficacious drugs. The results fit with clinical experience that the tetracyclines, (minocycline, doxycycline), sulfamethoxazole/trimethprim, lincosamides (clindamycin) and the macrolides (clarithromycin and azithromycin) achieve the greatest success.

Fortunately, there have been no reports showing any change in antibiotic resistance patterns regarding antibiotic susceptibility of the specific borrelial organisms in a given case.

Again, my personal preference is use of low-dose antibiotics such as doxycycline or minocycline.

Combinations of Antibiotics

Antibiotics used in combination are much more effective than used separately. Several antibiotics work together synergistically to improve the therapeutic effect against borreliosis. Examples of antibiotics that work well together when combined include: amoxicillin, metronidazole, clarithromycin or zithromycin, and co-trimoxazole. Co-trimoxazole works well in late stage Lyme disease, especially when given in combination with other antibiotics like amoxcilliln and/or a macrolide such as azithromycin. Earlier, one exception to antibiotic combinations was the use of metronidazole and the tetracyclines together. It is now believed that these two antibiotics do work well when combined. For example, there are reports of good result when doxycycline is used with flagyl.

Antibiotics that inhibit protein synthesis

Antibiotics that inhibit protein synthesis will also inhibit the production of BLPs by borrelia. This can be a useful tool for reducing the load of BLPs the body must deal with. Since the BLPs trigger the disease and the symptoms of borreliosis, reducing their production would be of significant benefit.

Tetracyclines

The tetracyclines including: tetracycline, doxycycline, minocycline; inhibit protein synthesis in bacteria. They do this by binding to the 30S unit of the bacterial ribosome. This binding blocks the bacteria's ability to synthesize proteins. Unfortunately, this binding is reversible. So, they must continued to be used until the bacteria die or are cleared from the body.

Macrolides and **Lincosamides**

The most widely used macrolides for LD are **azithromycin** and **clarithromycin** . These are relatively non-toxic antibiotics. Macrolides binds to the 50S ribosomal subunit and inhibits either peptidyl transferase activity or translocation of the growing peptide.

Clindamycin which is a lincosamide, has a similar mode of action as the macrolides.

Adjunct Anti-Microbial Therapies

Hyperthermia – Borrelia prefer temperatures below that of the body. Using hot showers, baths, or saunas at temperatures of up to 104 degrees F for 20-30 minutes daily to raise the body temperature is a helpful therapy for treating borreliosis. Borrelia species are especially sensitive to the combination of antibiotics and heat. Raising the body temperature also dilates the peripheral circulation and increases the permeability of the blood vessels throughout the body. These physiological changes assist in the delivery of antibiotics to all areas of the body increasing the amount of antibiotics able to penetrate and reach the borrelia. Hyperthermia improves the effect of antibiotic therapy by approximately 16 times.

Pulsing Electro-Magnetic Fields (pemfs) -- Recently, there has been a lot of progress in studying pemfs as a therapeutic modality for LD. This information is too numerous for me to list here so I'll refer to this message board that I follow and participate in where the members discuss pemfs design and therapies.

http://health.groups.yahoo.com/group/Magpulser/?yguid=159339971

Pemfs are very effective in pain and other symptom relief. I highly recommend it's use.

Here's a site that lists some of pemfs useful benefits:

http://www.magnetcure.com/reports.html

Rife Machine -- Rife machines are electronic devices that produce varying frequencies of energy (similar to a microwave, but at frequencies not harmful to the body). This energy penetrate the tissues of the body and cause the spiral-shaped Borrelia to resonate so much that the integrity of the bacteria is disrupted; weakening and even killing them. This is an effective means for killing borrelia and is supported by the production of herx reactions in Lyme patients after Rife treatments, while non-infected individuals don't experience the herx reactions when exposed to rife treatment. Rife machines are not approved by the FDA and are relatively expensive. A reasonable price for a good machine is about $1,000. Homemade ones can be much less expensive and just as effective. Here is a message board that I follow and participate in where the members discuss numerous alternative therapies including rifing: http://health.groups.yahoo.com/group/Lyme-and-rife/

Beck Electrification -- Low-voltage electrification is also useful for disrupting the integrity of microbes including borrelia. It may also enhance the ability of antibiotics to penetrate the cells of the body so they can reach the hidden intracellular borrelia. Herx reactions after treatment also substantiate the effect of this therapy.

Lauricidin -- The monoglyceride of lauric acid is called monolaurin. The concentrated form of monolaurin has potent antibacterial properties. While nontoxic and approved as a direct food additive by the FDA, monolaurin adversely affects bacteria, yeast, fungi, and enveloped viruses. It damages the cell membrane of pathogenic bacteria.

www.lauricidin.com

Colloidal Silver -- Colloidal silver therapy for Lyme disease appears to be significantly effective with many individuals that have used it. There is much controversy regarding this therapy. Argyria, is the name of the condition in which the skin turns bluish-gray from silver. This is very rare and is linked to the use of silver salts and not pure colloidal silver. It appears to be important to use pure colloidal silver. The size of the colloidal particles is also important. The smaller the particles size of CS the better the bioavailability, the stronger the anti-microbial effect and the safer to use.

Cat's Claw / Samento / Saventero – A Peruvian herb *(Uncaria tomentosa)* is quickly becoming a rising star within the LD community. It appears to have both anti-microbial and anti-inflammatory properties both of which are significantly effective. Tolerance is a question for some. It can cause anxiety, insomnia, and irritability in certain individuals. Adjusting the dose appropriately can control this side-effect. Even at very low doses it seems to be beneficial.

Artemisinin – Is the active constituent of the herb *(Artemesia annua)* . Tea made from this herb has been used in Asia to successfully treat malaria. Artemisinin (pronounced art-ee-MISS-in-in) is the preferred antimalarial therapy. It is also being used to treat cancer in veterinary medicine and is an effective anti-parasite and anti-microbial treatment.

Artemisinin also seems effective in treating LD. There are many testimonials from users claiming significant benefits.

Goldenseal root – The best brand out there is may be Eclectic Institute's freeze-dried goldenseal root.

Teasel – Another effective herbal therapy.

Olive Leaf Extract – (Oleuropein) Must be taken at high doses though. Take 1500 – 2000 mg three times per day.

Garlic – Antimicrobial and helps reduce blood clotting. Use fresh garlic or freeze-dried supplements.

European (or Hungarian) Mistletoe – There is a little information that this may be beneficial for LD as an antimicrobial.

Peroxide – Is a potent antiseptic but I'm concerned about the safety of oral and IV administration.

Ozone – Similar concern as with peroxide.

Controlling Inflammation and the Immune Dysfunction

Inflammation is a major part of any disease, but none more complex and involved than with Lyme disease. Borrelia BLPs that has disseminated throughout the body will trigger inflammation in any and all systems, organs and tissues that it resides in. This is how Lyme can cause so many different symptoms and mimic so many disorders. Dealing with this complex inflammation is a major part of managing Lyme disease. Controlling inflammation begins with a

well balanced **low-carb diet**
. Why? Basically because sugars and starch promote inflammation by altering the body's metabolism of fatty acids. I explain this in depth in my writings on insulin resistance and the inflammatory syndrome (IRIS). The low-sugar/starch diet also reduces the chance for yeast infections to become established. Nystatin (an antimicrobial for yeast) can be taken orally to also prevent yeast growth. (If you would like any more information on the low-carb diet, please refer to my book, "Dr. Taylor's Remedi for Healthy Living." You can order it on my website at www.myremedi.com . or toll free at 877-736-7348.)

There are several dietary supplements that help reduce inflammation *:* **MSM** , Salmon oil, **Aflamend™** , and **Lyprinol ®** work well. *MSM*
, Methylsulfonylmenthane (MSM) is a very safe supplement that is used to reduce inflammation without the risk of any side effects. It is inexpensive, but therapeutic doses for an adult are high. *Salmon oil* is abundantly rich in the Omega-3 fatty acids. The fatty acids EPA and DHA that are found in Salmon oil help reduce inflammation and have many other health benefits. *Aflamend*
™ is a blend of herbal extracts I designed to help reduce inflammation. Aflamend™ augments the health benefits of Dr. Taylor's " Remedi for Healthy Living
" program by improving insulin sensitivity and enhancing a healthy fatty acid metabolism, both of which reduce chronic inflammation.

Lyprinol, ®
a specific stabilized extract of green lipped mussels, is a relatively new supplement for inflammation. Early feedback of Lyprinol ®
use is reporting several beneficial results for people with many stubborn inflammatory disorders. This product is a unique fatty acid that is an effective tumor necrosis factor (TNF), lipoxygenase and (LOX) inhibitor. Its potency to reduce inflammation is approximately 200 times stronger than fish oil. It is also an effective MMP-9 inhibitor thus it helps reduce the MMPs that digest the extracellular matrix proteins of the body.

Other inhibitors of TNF include Boswellia, Quercetin, Cat's Claw, Turmeric, NAC (N-acetyl-cysteine).

Nettle also has the ability to inhibit the release of MMP triggered by BLPs.

Cherry Fruit extract, Cetyl Myristoleate, help inflammation problems.

Other dietary supplements helpful for Lyme patients include: bovine colostrums, lycopene and DHEA to improve growth hormone and other hormone deficiencies, the minerals magnesium and potassium, vitamin C, vitamin E (mixed tocopherols), and a good multi-vitamin that contains no retinyl palmitate form of vitamin A. Vitamin B12 helps deal with neuropathies.
It can be taken sublingual (under the tongue) daily or preferably by subcutaneous weekly injections. Other supplements that will help to reduce inflammation and support the immune system include Borage and/or Evening Primrose oil, and DMAE. DMAE (dimethylaminoethanol) helps with fatigue caused by Lyme disease. Astragalus is a herb that is used for immune support.

Protecting the Nervous System from Neurotoxins

It is important to protect the peripheral and central nervous system from the toxins produced by borrelia. The following is a list of dietary supplements that are effective for this:

SAMe : S-adenosyl-L-methionine: The beneficial effects of SAMe supplementation are extensive because this nutrient is involved in so many metabolic processes, including its role in serving to detoxify cell membranes and synthesize neurotransmitters. From acting as an antioxidant to raising serotonin levels in the brain, SAMe is one of the most important compounds to come to the market. Studies on the use of SAMe in maintaining normal joint function are also promising.

Phosphadityl choline: Take 1 tablespoon of lecithin with each meal.

DMAE (dimethylaminoethanol) is a precursor to acetyl choline and has many benefits for LD therapy.

B-vitamins: LD patients need high doses of B-vitamins, especially B-6, **B-12** , and folic acid.

Anti-oxidant that are effective at protecting the nervous system include: pycnogenol, **grape seed extract** , bilberry, and **alpha lipoic acid** .

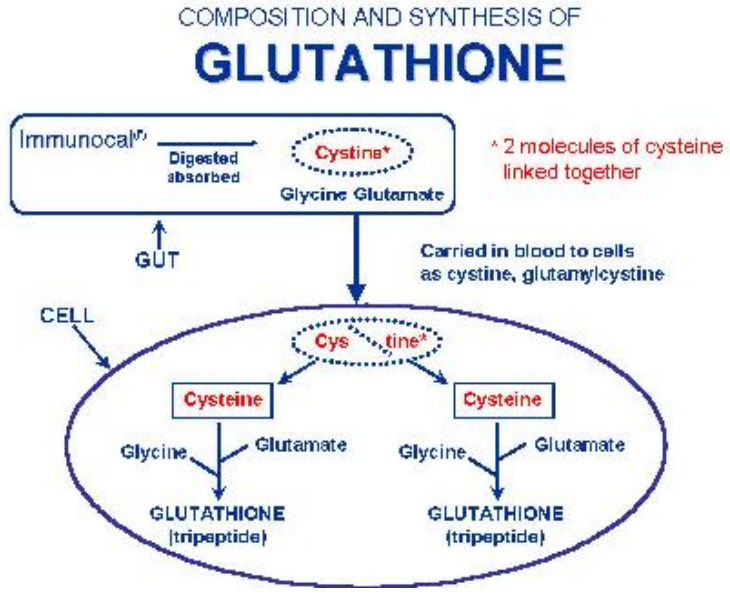
Detoxifying and Excreting the Toxins of Borrelia

Borrelia produce numerous toxic BLPs. These toxins are important because they trigger many harmful responses in the body including the inflammation that is damaging to healthy tissue, and cause the dysfunction of the immune system. These toxins are fat-soluble lipoproteins and are very difficult to rid the body of. The body normally detoxifies fat-soluble substances in the liver and excretes them from the bile. Unfortunately, the toxins appear to be reabsorbed from the gut and circulate back into the body.

Treatments designed to aid the body in eliminating these toxins is a very important part of a complete and comprehensive Lyme disease therapy.

Glutathione is the most important detoxifying agent in the body to get rid of undesirable toxins such as BLPs produced by borrelia. It forms a soluble compound with the toxin that can then be excreted through the urine or the gut. The liver and kidneys contain high levels of glutathione as they have the greatest exposure to toxins. The lungs are also rich in glutathione partly for the same reason. Glutathione is a small molecule found in almost every cell. It cannot enter most cells directly. Instead glutathione must be synthesized inside the cell from its three constituent amino acids: glycine, glutamate and cysteine. The rate at which glutathione can be made depends on the availability of cysteine, which is relatively scarce in foodstuffs. Furthermore, the cysteine molecule has a sulfur-containing portion which gives the whole glutathione molecule its 'biochemical activity', i.e. its ability to carry out its vitally important functions. Glutathione is the major antioxidant produced by the cell, protecting it from 'free radicals' ('oxygen radicals', 'oxyradicals'). These highly reactive substances, if left unchecked, will damage or destroy key cell components (e.g. membranes, DNA) in microseconds. Oxyradicals are generated in the many thousand mitochondria located inside each cell, where nutrients like glucose are burnt using oxygen to make energy. (Mitochondria can be thought of as the batteries that provide the power for the cells to operate). High levels of oxyradicals are also generated from inflammation or immune dysfunctions such as in chronic LD.

Thus, glutathione is required in many of the intricate steps needed to carry out an immune response. For example, it is needed for the lymphocytes to multiply in order to develop a strong immune response, and for 'killer' lymphocytes to be able to kill undesirable cells such as cancer cells or virally infected cells. The importance of glutathione cannot be overstated. It has multiple roles as indicated and, indeed, as one examines each system or organ more closely, the necessity for glutathione becomes increasingly evident. Glutathione values decline with chronic disease and age. Higher values in older people are seen to correlate with better health, underscoring the importance of this remarkable substance for maintaining a healthy, well-functioning body.



The two dietary supplements, undenatured whey proteins and NAC (N-acetyl-cysteine), are the best cost effective way to increases the production of intracellular glutathione.

The biliary system gives us a chance to trap the toxins while they are in the gut. Substances that trap fat-soluble compounds can be taken orally to do this. Care must be taken not to use these chronically because they can have side effects and reduce the absorption of healthy fat soluble vitamins and nutrients.

Chlorella

Very effective detoxifier

Stimulates the immune system

Contains growth factors that stimulate the regeneration of damaged tissues.

Chitosan: a shellfish fiber that traps lipids.

Bentonite: is a clay-like substance that attracts lipophilic compounds

Apple Pectin:

Cholestyramine (Questran or Cholistad): a prescription that traps lipophilic compounds. Cholestyramine can cause constipation as a side effect.

Milk Thistle : Is probably the best herb that helps the liver detoxify and excrete bile.

Exercise & Sauna: increases the production of bile and mobilizes lipids. Heat therapy is a very effective detoxifying therapy.

Scrubing skin with sponges or brushes enhances the skins ability to remove toxins.

Using fiber (**psyllium**) to scrub the GI tract removes dead epithelial cells that contain toxins.

NAC (N-acetyl-cysteine): The best supplement that increases the production of glutathione, which is used in detoxification.

Taurine : An amino acid that aids in detoxification by providing a good source of sulfur.

Immune Modulation

Therapies to improve or repair the dysfunction in the immune system are important remedies when dealing with chronic disorders such as LD. The hope of this type of therapy is to tilt the balance of the immune system in favor of defending the body from chronic borrelia infection.

Tick Protection and Prevention

Repellant sprays are a useful tool to reduce exposure to ticks. Products that contain DEET are good tick repellents. They do not kill the tick and are not 100% effective in discouraging a tick from feeding on you. Products like Permanone contain premethrin, and are known to kill ticks. However, they are not designed to be sprayed on the skin. Premethrin can be sprayed on clothing. Once it is dry it is assumed to be safe to touch. Pest resistant clothing can offer some protection. Ticks are anti-gravitational, i.e., they generally seek the highest point on the body. If they get on the body below the clothes line, they will travel up and die once they come in contact with premethrin treated clothing. Note: If the tick meets resistance on its journey to the head, it will stop and feed at that point.

Tick Removal

If an attached tick is found on your body, remove it carefully with fine tweezers. Grab the tick as closely to the skin as possible. *Do not squeeze the body; do not apply Vaseline; do not touch the tick with a burnt match; and do not clean it with alcohol while the tick is attached.*
Any of these actions could cause transmission of Lyme disease by causing the tick to regurgitate the borrelia bacteria that live in their gut into the skin where it is attached. Ticks can be identified and tested for the bacteria. However, Borrelia frequently cannot be grown in the laboratory due to its fastidious nature and so nothing shows up. It's still important to save the tick for identification as a possible vector. Place the tick in a glass or plastic vial with a few blades of moist grass or a moistened cotton ball. A clean pill vile is good. If none is available, use a Ziploc storage bag as a temporary container.

Stop It Before It Starts

I strongly recommend prophylactic treatment when a person is bitten by a tick, especially if the tick has been attached over 24 hours. Since there isn't enough evidence on either side to prove or disprove this theory, the final decision is up to the individual and the treating physician. The recommended prophylactic treatment is 4-6 weeks of an oral antibiotic. 100 mg doxycycline tid (three times per day) or 1000 mg amoxicillin tid for prevention for at least

6 weeks is recommended.

Vaccine

As of March, 2002, the Lymerix ®
vaccine is no longer available. In December 1999, a class action suit was filed against SmithKline Beecham, the manufacturers of the LYMErix ® vaccine.
The complaint alleges the manufacturer failed to warn doctors and the general public that nearly 30% of the general population was genetically pre-disposed to a degenerative autoimmune syndrome, including chronic arthritis, which the lawsuit says, is triggered by the OSP-A contents of the vaccine. We know that the OSP-A contains many of the toxic BLPs that trigger LD. Many private lawsuits have also been filed from individuals who received the vaccine and are now disabled with chronic arthritis. So, this means that a safe vaccine is not yet available to the public.

Animals

Lyme disease can affect individual pets differently. Some animals may display no symptoms. Other animals may develop fever, loss of appetite, painful joints, lethargy, and vomiting. If left untreated, the spirochete may damage the eyes, heart, kidneys, and nervous system. Lyme disease has been diagnosed in dogs, cats, horses, goats, and cattle. Other species may also be at risk.

Infected dogs may be lethargic, have a poor appetite, or a fever. Dogs may also experience lameness shifting from one joint to another, fatigue, kidney damage or failure, heart disorders, or neurologic involvement (e.g. aggression, confusion, overeating, and seizures). Dogs can also be infected with the Lyme bacterium but not exhibit any noticeable symptoms. Transplacental transmission has occurred in dogs.

Many cattle do not display signs of Lyme disease; those that do may have lameness, painful or swollen joints, fever, or weight loss. A skin rash may be present on the udder of infected cows. "Bb" has been found to exist in urine and colostrum of infected cattle; therefore, the possibility of transmission between cows should be considered. The Lyme bacterium has also been found in blood, milk, synovial fluid and spontaneously aborted fetal tissue. "Bb" can survive in frozen milk, but is killed during pasteurization due to its sensitivity to heat.

Infected horses generally do not have a fever, but may have lame or stiff joints, laminitis, depression, or refusal to eat. This bacterial infection may be a cause of moon blindness or loss of vision. There have been reports of spontaneous abortion and encephalitis in horses infected with "Bb". Neurologic signs include head tilt, difficulty swallowing, or aimless wandering. Transplacental transmission occurs. Colts born to infected mares have displayed birth defects. Many horses may be infected with the spirochete, but display no symptoms.

Cats may show lameness, fever, loss of appetite, fatigue, eye damage, unusual breathing, or heart involvement. However, many cats do not show noticeable symptoms, despite being infected.

Humans and monkeys are more susceptible to neuroborreliosis than other animals.

What Can You Do?

First of all, prevention is the best cure. If you have to be outside, use insect repellent with deet. Take a hot shower as soon as you can and check, in your hair especially, for any ticks. Since it is believed that a tick needs to be on your skin for at least 24 hours before spreading the disease, this prevention could save you in the long run. Strongly consider prophylactic antibiotics when bitten by a tick.

If you are suffering with a number of different symptoms and you aren't quite sure what you have, don't rule out Lyme disease. This disease is called "the great imitator" and you don't need a rash on your body to have it. If these symptoms have been going on for a while and you think there is a chance, I would strongly recommend contacting a Lyme disease specialist. One specialist that I know is Dr. Charles Crist in Springfield, MO (417) 886-8389. Here's his website: www.drcharlescrist.com
. For more information about doctors that have worked with LD patients in the state of Iowa, you can contact Judy Weeg who is with the Central Iowa Lyme Disesase Association at (515) 388-1401.

There is Hope

You can find great support at thes websites: www.MarshallProtocol.com and www.lymenet.org

Go to the flash discussion group at lymenet. There are many helpful members of this message group. It's a great site

for support and helpful information.

While many in the medical community are "Lyme ignorant," there are still a few knowledgeable doctors that can be found in Iowa and near the borders. Some people that have been diagnosed with Lyme disease have been given the right treatment for a long enough period to fully recover and are cured. Others carry it and battle with it the rest of their lives. There are others, however, who have been misdiagnosed and mistreated who continue to live with the problems of Lyme disease and they never fully recover. In 1999, two Iowans in their 30's died unexpectedly from the unknown complications of Lyme disease. As the word spreads about the complex nature of this disease, the hope is that more doctors will begin to learn about Lyme, and will take the actions necessary to fight this disease. It is critical that the public and the medical community are made aware of the true prevalence and dangers of borreliosis. Until this gap of ignorance is filled, many unfortunate individuals will suffer needlessly with Lyme disease.