Release Notes 2021-06-01: : This is close enough to a manuscript to release as it makes the intended points in an informal style with enough references for a short work addressed to an audience familiar with the topic. Recent results feeding heartworm positive and pregnant dogs, if I have accurately described their condition, with supplements including significant vitamin K have been very encouraging or at least not harmful. An explanation for this may be useful to a wider audience and certainly critical discussion could be useful given all the paradoxes in this important area. The phrase " true enough to be good" is used in the text. It derives from " too good to true" and a popular jumbled version " too true to be good" with many meanings but in physics motivating the need for approximations to make an exact mathematical expression comprehensible. It is always disconcerting to create a curve with few numbers behind it but it appears to fill a need until a critic can demonstrate otherwise. Caveat Emptor.**I'm not a doctor or a vet and this is not medical advice or advocacy of any specific acts. See fuller disclaimers at the end along with information for citing.**

A Proposed Qualitative Non-monotonic Paradox Resolving Actvitiy-Coagulability Curve for Vitamin K

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(Dated: June 1, 2021)

This letter describes the synthesis of a qualitative curve illustrating non-monotonic effects of vitamin K on coagulation that harmonizes paradoxical observations. The curve relates two imprecise concepts- the activity of reduced vitamin K and coagulability state- that are common topics in the literature. The proposed relationship is no better than an idealized illustration of a more complicated situation that requires experimental validation but , if "true enough to be good" , by resolving paradoxes it may improve understanding and motivate better disease interventions. It is used to introduce a larger issue with vitamin K replacing anti-coagulants in some typical indications [9] [10].

Despite some successes, concerns already exist about inappropriate use of anti-coagulants [23]. In almost all cases of coagulopathy or inappropriate coagulation, vitamin K is assumed to be a problem rather than a solution even when bleeding is a component of the problem and the reader is likely familiar with this literature. There are limited cues to the contrary however. A 1987 paper suggested that vitamin K deficiency was responsible for coagulopathy in a group of critically ill patients [2]. A 1998 work found subtle vitamin K deficiencies in hospitalized patients [6] and a 2004 work warned that vitamin K deficiencies are common in ICU patients [21]. While not generally appreciated, these observations may also explain paradoxes with warfarin and suggest a clinical role for the coagulation moderating vitamin K dependent proteins(VKDP). It may be that their observations are more broadly applicable to larger patient groups as described here.

This work uses just a few VKDP's for which literature was quickly found and that require vitamin K to form Gla domains for maturation. The four proteins considered here are prothrombin, factor X, protein S, and protein Z. The first two tend to promote coagulation while the latter tend to moderate it. The quantity, fate, and maturity of each may be differently effected as reduced vitamin K becomes less available. Prothrombin from dicoumarol treated bovine did demonstrate a non-random undercarboxylation [7] with clotting activity related to amount of carboxylation [8]. Another work found that partially carboxylated protein S could be held in human vascular smooth muscle cells until vitamin K was restored to complete carboxylation [3]. One interesting work compared the output of FX and protein Z as a function of warfarin with and without vitamin K [24] demonstrating protein Z may be more sensitive than FX to inhibition. The function of most of these proteins remains partially unknown and protein Z seems to have an expanding role. To illustrate some possible other issues with vitamin K depletion , in 2013 protein Z was described as,

> ...lack[ing] any enzymatic activity. Instead, PZ acts as a cofactor for the inhibition of factor Xa through the serpin PZ-dependent protease inhibitor (ZPI). PZ deficiency is associated with a procoagulant state, highlighted by excessive FXa secretion and thrombin production, and is linked with several thrombotic disorders, including arterial vascular and venous thromboembolic diseases. A role for the PZ-ZPI complex in the regulation of physiological pregnancy has been demonstrated, highlighted by the progressive elevation in PZ levels in the first trimester of gestation, which then steadily decline toward delivery. An association between altered plasma PZ concentrations and adverse pregnancy outcomes (recurrent miscarriage,

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stillbirth, preeclampsia, intrauterine growth restriction, and placental abruption) has been reported. The mechanism by which PZ deficiency leads to adverse pregnancy outcomes is not clear, but it is multifactorial. It may be attributed to the anti-PZ IgG and IgM autoantibodies, which apparently act independently of classical antiphospholipid antibodies (lupus anticoagulant, anticardiolipin, and anti-2-glycoprotein I antibodies). PZ deficiency has also been reported to be constitutional, and a number of variants in the PROZ (PZ) gene and SERPINA10 (ZPI) gene are linked with specific adverse pregnancy complications [1].

Overall, the coagulation nuclei and the lifecycles need to be more fully considered as part of a coagulability state. VKDP's also serve to regulate some of these such as NET's. Protein C, also a VKDP that moderates coagulation, is noted as antagonistic towards histone enhanced thrombin generation [22], as well as NET's in VT [18] and other conditions [5]. Very recently, NET's were implicated in bleeding problems when using tPA to remove clots from mice with ischemic strokes [26] which suggests the importance of the nuclei discussed here. As fibrin is observed to reduce NET formation [27], some fibrin formation is likely beneficial.

Some hints of non-monotonic effects arise from clinical and lab work that may be well known and accepted but not fully appreciated. For example, initial use of vitamin K antagonists creates a temporary pro-coagulant period [4] . In chronic usage, the therapeutic window appears to be small and dosage control is difficult. Confusing results were obtained with anticoagulation in covid-19 including a failure [28] and a "plasmin paradox" has been described with fibrinolytics leading the authors to consider inhibition of fibrinolysis although the promiscuous nature of plasmin [19] creates a different set of issues than on the creation side. In some indications it is becoming clear that anti-clotting makes things worse contrary to expectations. Warfarin for idiopathic pulmonary fibrosis, for example, appeared to be ineffective or counter productive in a trial that was terminated early for safety and futility concerns [20].

Precise quantitative modelling would help but it is difficult as the coagulation system is probably only partially known and empirical studies complicated by error sensitivity with a large number of parameters and positive feedback loops. The vitamin K system also has confusing interactions with systems of variable relationship to coagulation. However, a conceptual simplified curve ,Fig. 1 , that is consistent with known functions of vitamin K makes it possible to rationalize clinical and biochemical paradoxes. The curve relates vitamin K activity(A_{vK}) to a generalized coagulability state. Note that "activity" is a colloquial term but includes ability to cycle vitamin K back to a fully reduced state and is not just the quantity of vitamin K. Both axes are arbitrary but scaled such that "1" is normal for both quantities. A "safe" range of coagulability is arbitrarily taken as $.9-1.1$ for illustration. Coagulability above suggests clotting risks while much below fails to stop bleeding. A hypercoagulable state exists for A_{vK}between about .084 and .683 while bleeding risk is too high if A_{vK}falls below .077.

A hypercoagulable state can then be corrected with anti-coagulants to bring A_{vK}into a narrow range around .08. Alternatively, it can be corrected and all the functions of vitamin K preserved by increasing A_{vK}above .684. This may involve more vitamin K, faster cycling, or fixing some other pathway blockage.

The lower limit should generally not be controversial although the exact form may be of some significance. Popular wisdom is that more vitamin K leads to more coagulation and that too much is bad. However, this is not easy to reconcile with the generally accepted safety of high doses for healthy people. Further, experience with heartworm positive dogs described later is favorable.

A typical disease may start from healthy, progress to increasingly more pro-coagulant, and then finally to bleeding but not uniformly. This makes reasonable sense then by following this curve from normalized vitamin K activity around 1 to lower values first leading to too much coagulation and then finally more bleeds towards the origin. Correcting hypercoagulability, where the coagulability state is much greater than 1, with vitamin K antagonists requires then maintaining a narrow range as this curve passes through "1" around $A_{vK}=$ .08 with a large slope which could explain the difficulty controlling warfarin therapy. Alternatively, saturating the vitamin K system will producing acceptable results at some minimum activity up to practically unlimited high doses. While this would most clearly apply to vitamin K antagonists, many coagulopathies may have vitamin K status as a contributing factor and attempts to adjust some other level will not likely be as successful as fixing A_{vK}particularly as the coagulation system may be needed to respond to the disease and vitamin K may be needed for other functions. There may be a concern in going from the "warfarin optimum" around .08 to the more stable optimum above about 1 and traversing the hypercoagulable range. However, the system seems possibly adapted to react well to supplementation and is likely not worse than the observed pro-coagulant state when first starting warfarin. For example, protein S remains sequestered until fully carboxylated and additional vitamin K could release a lot of this quickly compared to say the incremental prothrombin output. This curve does not capture dynamics but what is known is supportive of "safe passage" to the more stable state being feasible.

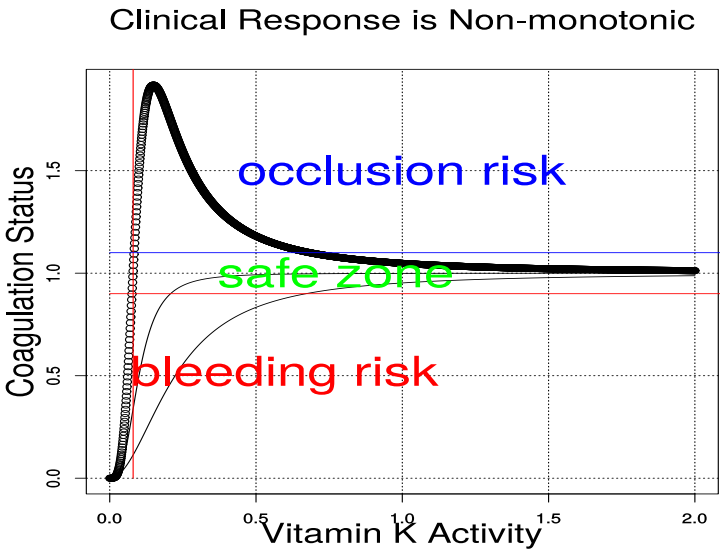

FIG. 1: Hypothetical illustration of competition that can produce non-monotonic coagulation state. Coagulation state is the thick curve, which may be a ratio of two monotonic but opposing quantities, similar to the lighter curves(no y-axis given for these) , which may something like prothrombin and protein S maturation. Both the "global vitamin status" and " coagulation state" are poorly defined ideas but routinely discussed so the quantitative approach was chosen a bit arbitrarily. Non-monotonic response curves are not always appreciated but in reality few curves are infinitely monotonic. During a nuclei producing disease, vitamin K cycling demands can increase pushing the vitamin K status to the left rationalizing a common clinical course of pathological clotting followed by bleeding.

Its interesting to note that vitamin K was first discovered during feeding experiments with animals, actually chicks [25], so there is some precedent for this simple approach to discover interesting practical results. At least three heartworm positive dogs have been given significant amounts of vitamin K (10mg-15mg/day intermittently with weights in the 5-30kg range) along with other nutrients. The first documented case, Happy [11] [14] [15], was monitored by her cough and general physical activity. While cough did increase after a large initial decrease and there was some occasional lack of interest in walking, it is not likely these were cardiovascular related. She eventually was found heartworm and murmur free and continues to do well today although she coughs or honks sometimes . A more recent dog, Andy[13], arrived with little interest in exercise but his exercise tolerance improved well after Diroban treatment. He did however experience significant peritoneal fluid accumulation which did not seem to impact his exercise tolerance-indeed, he could walk a quarter mile or so while grossly inflated from the fluid without significant effort. He was lost to follow up but apparently eventually resolved and he was given with sildenafil after being diagnosed with PAH. His symptoms appear (to me) more consistent with aquaporin activation or a transient thyroid issue (and his only thyroid test here demonstrated a total T4 low enough to treat, .8 versus 1-4 $\mu g/dl$). While details are unclear, his last known status was normal and "running on the beach". A third dog, Brownie [17] , has yet to be treated for heartworm but delivered 12 puppies of which 9 have survived for 3 months with the only known problems being 2 eyelid deformities. She received the vitamin K diet during most of her term and the puppies received it too once old enough to eat solid food. Diet and note data are available in MUQED format with varying degrees of completeness [12] [16]. Other nutrients may contribute to or confound results including taurine, potassium chloride, lecithin, niacin, and amino acids.

In the absence of more quantitative or less ambiguous data, hopefully this curve is "true enough to be good" and will create a better conception for the effects of varied levels of reduced vitamin K activity and begin to resolve paradoxes and dilemmas involving vitamin K and coagulation.

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1. SUPPLEMENTAL INFORMATION

1.1. Computer Code

Acknowledgments

1. Pubmed eutils facilities and the basic research it provides.
2. Free software including Linux, R, LaTex etc.
3. Thanks everyone who contributed incidental support.

Appendix A: Statement of Conflicts

No specific funding was used in this effort and there are no relationships with others that could create a conflict of interest. I would like to develop these ideas further and have obvious bias towards making them appear successful. Barbara Cade, the dog owner, has worked in the pet food industry but this does not likely create a conflict. We have no interest in the makers of any of the products named in this work.

Appendix B: About the Authors and Facility

This work was performed at a dog rescue run by Barbara Cade and housed in rural Georgia. The author of this report ,Mike Marchywka, has a background in electrical engineering and has done extensive research using free online literature sources. I hope to find additional people interested in critically examining the results and verify that they can be reproduced effectively to treat other dogs.

Appendix C: Symbols, Abbreviations and Colloquialisms

TERM	definition and meaning
FX	Factor X or 10
ICU	Intensive Care Unit
NET	Neutrophil Extracellular Trap
PAH	Pulmonary Arterial Hypertension
PROZ	Protein Z
PZ	Protein Z
PZ-ZPI	Protein Z : PZ-dependent protease inhibitor complex
VKDP	Vitamin K Dependent Protein
VT	Venous Thromboembolism

Appendix D: General caveats and disclaimer

This document was created in the hope it will be interesting to someone including me by providing information about some topic that may include personal experience or a literature review or description of a speculative theory or idea. There is no assurance that the content of this work will be useful for any paricular purpose.

All statements in this document were true to the best of my knowledge at the time they were made and every attempt is made to assure they are not misleading or confusing. However, information provided by others and observations that can be manipulated by unknown causes may be misleading. Any use of this information should be preceded by validation including replication where feasible. Errors may enter into the final work at every step from conception and research to final editing.

Documents labelled "NOTES" or "not public" contain substantial informal or speculative content that may be terse and poorly edited or even sarcastic or profane. Documents labelled as "public" have generally been edited to be more coherent but probably have not been reviewed or proof read.

Generally non-public documents are labelled as such to avoid confusion and embarassment and should be read with that understanding. ‘

Appendix E: Citing this as a tech report or white paper

Note: This is mostly manually entered and not assured to be error free.
This is tech report MJM-2021-004.

Version	Date	Comments
0.01	2021-05-30	Create from empty.tex template
0.9	2021-06-01	Draft Manuscript
-	June 1, 2021	version 0.90 MJM-2021-004
1.0	20xx-xx-xx	First revision for distribution

Released versions,
Build script needs to include empty releases.tex

Version	Date	URL
0.90	2021-06-01	https://www.academia.edu/attachments/67479547/download_file
0.90	2021-06-01	https://www.researchgate.net/publication/352020800_A_Proposed_Qualitative_Non-monotonic_Paradox_Resol
0.90	2021-06-01	https://www.linkedin.com/posts/marchywka_vitamin-k-and-coagulation-activity-6805481117047681024-3ez_

@TECHREPORT{mmarchywka-MJM-2021-004-0.90 ,
AUTHOR = {M.J. Marchywka},
TITLE = { A Proposed Qualitative Non-monotonic Paradox Resolving Actvitiy-Coagulability Curve for Vitamin K},
NUMBER = {MJM-2021-004},
VERSION = {0.90 June 1, 2021 DRAFT INFORMAL MANUSCRIPT },
INSTITUTION = { not institutionalized , independent},
ADDRESS = {306 Charles Cox , Canton GA 30115},
NOTE = {Version 0.90 , may change signficantly if less than 1.00 },
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DAY = {1},
MONTH = {6},
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PAGES = { 7 },
CONTACT = {marchywka@hotmail.com},
FILENAME = {vkcurve}
}

Supporting files. Note that some dates,sizes, and md5's will change as this is rebuilt.

This really needs to include the data analysis code but right now it is auto generated picking up things from prior build in many cases

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31050 Jul 21 2011 /usr/share/texlive/texmf-dist/bibtex/bst/urlbst/plainurl.bst
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306133 Jun 1 09:13 vkcurve.pdf ce8e757238984945450b95c6cbcd9b1e
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