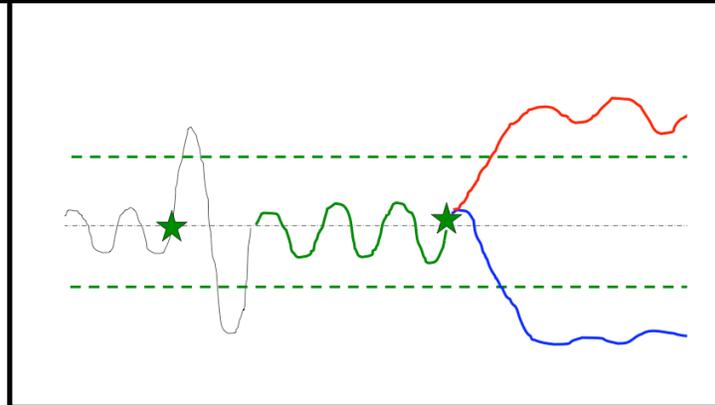


## Phases in the Origins of Chronic Symptoms and Chronic Illness



Time across the lifespan – from conception forward →

- 1. Predisposition to a symptom or chronic illness (ie: lupus, diabetes, chronic fatigue, bipolar,..)**
  - affects risk by imprinting, conditioning & predisposing the nervous system to regulate in certain ways<sup>4</sup>
  - appears to occur in early life such as during pregnancy, birth, and in childhood<sup>5</sup>
  - is influenced by bonding and attachment relationships, through which nervous systems learn to regulate<sup>6 7</sup>
  - is shaped by *trauma*<sup>8</sup>, which reduces sense of safety and trains the nervous system to regulate defensively<sup>9</sup>
  - is influenced by timing: developing organs are shaped, and therefore most influenced, by environmental factors including life experiences such as *parenting care*, which turn genes on/off<sup>10 11</sup> and shape patterns of regulation; *early life events*, which can affect stress responses for life<sup>7 12</sup>, risk for type 1 diabetes<sup>13-15</sup>, MS<sup>16</sup>, Parkinson's<sup>17 18</sup>, and *trauma*, which may play a role in diabetes<sup>1 12</sup>, Parkinson's<sup>19-21</sup>, & other diseases
- 2. Latency Period: A period predating diagnosis**, during which conditioned responses grow and strengthen
  - a classic example is posttraumatic stress, in which symptoms occur days, months or years after trauma<sup>22</sup>
  - conditioning is strengthened by events that stress the system, such as trauma, increasingly mild and seemingly unrelated events that are stressful, and other environmental factors (pollution, infections, etc...)<sup>4</sup>
  - a greater intensity or frequency of stressors favors certain patterns of “wiring” and shorter latency periods
  - supportive (resourcing) life experiences serve as buffers that may lengthen the latency period, delay the onset of illness, or protect the organism and prevent illness<sup>4</sup>
  - latency periods may last 7 yrs in lupus<sup>23</sup>; 10 years + in type 1 diabetes<sup>24 25</sup>; 30 years in Alzheimer's<sup>26</sup>; and may begin as early as adolescence in Parkinson's<sup>27</sup> or earlier<sup>18</sup>
- 2. Unmasking or ‘Reinstatement’ of the Conditioned Pattern: Diagnosis**
  - a final event (which may seem small) makes the conditioned response visible, dominant, & symptomatic<sup>28</sup>
- 4. Symptom Onset: occurring days, months, years following the “final stressor”**
  - clinical symptoms are unmasked, representing changes in nervous system patterns
  - symptoms represent an intelligent, albeit magnified and prolonged “survival strategy” or defense response<sup>4</sup>
  - the more severe the symptom, the more intense the preceding and ongoing *perceptions* of threat may be<sup>4</sup>, and the more severe the state of undischarged arousal (see trauma literature<sup>1 4 8 29</sup>)
  - type of symptom or illness is influenced by the timing of initial predisposing events (critical period)<sup>7</sup>
- 5. Triggers**
  - symptoms vary according to influence of past experiences (buffers and stressors) unique to each person<sup>4</sup>
  - triggers can exacerbate symptoms; identifying triggers may help predict and reduce exacerbations

## References for "Phases in the Origins of Chronic Symptoms and Chronic Illness"

1. Mead VP. A new model for understanding the role of environmental factors in the origins of chronic illness: a case study of type 1 diabetes mellitus. *Medical Hypotheses* 2004;63:1035-1046.
2. Mead VP. Somatic psychology theory and the origins of chronic illness: a case study of type 1 diabetes [Master's Thesis]. Naropa University, 2003.
3. visit [www.veroniquemead.com](http://www.veroniquemead.com). for additional information on trauma and early life events (library), nervous system regulation (theory), upcoming talks in Boulder (events), etc.
4. Scaer RC. *The body bears the burden: trauma, dissociation, and disease*. New York: Haworth Medical, 2001.
5. Dahlquist G. The aetiology of type 1 diabetes: an epidemiological perspective. *Acta Paediatr Suppl* 1998;425:5-10.
6. Klaus MH, Kennell JH. *Maternal-infant bonding*. St. Louis: Mosby, 1976.
7. Schore AN. *Affect regulation and the origin of the self: the neurobiology of emotional development*. Hillsdale, NJ: Lawrence Erlbaum, 1994.
8. Scaer R. *The trauma spectrum: Hidden wounds and human resiliency*. New York: W.W. Norton, 2005.
9. Scaer RC. The neurophysiology of dissociation and chronic disease. *Appl Psychophysiol Biofeedback* 2001;26(1):73-91.
10. Meaney MJ. Maternal care, gene expression, and the transmission of individual differences in stress reactivity across generations. *Annu Rev Neurosci* 2001;24:1161-92.
11. Weaver IC, Cervoni N, Champagne FA, D'Alessio AC, Sharma S, Seckl JR, et al. Epigenetic programming by maternal behavior. *Nat Neurosci* 2004;7(8):847-54.
12. Nathanielsz P. *Life in the womb*. Ithaca, NY: Promethean, 1999.
13. Dahlquist G, Kallen B. Maternal-child blood group incompatibility and other perinatal events increase the risk for early-onset type 1 (insulin-dependent) diabetes mellitus. *Diabetologia* 1992;35(7):671-675.
14. Patterson CC, Carson DJ, Hadden DR, Waugh NR, Cole SK. A case-control investigation of perinatal risk factors for childhood IDDM in northern Ireland and Scotland. *Diabetes Care* 1994;17(5):376-381.
15. Leslie DG, Elliot RB. Early environmental events as a cause of IDDM. *Diabetes* 1994;43:843-850.
16. Maser C. [The perinatal period of multiple sclerosis patients]. *Schweiz Med Wochenschr* 1969;99(50):1824-6.
17. Singer C, Weiner WJ. Could Parkinson's disease follow intra-uterine influenza? A speculative hypothesis [letter; comment]. *J Neurol Neurosurg Psychiatry* 1989;52(7):931.
18. Gibb WRG, Lees AJ. The relevance of the Lewy body to the pathogenesis of idiopathic Parkinson's disease. *Journal of Neurology* 1988;51:745-752.
19. Maher NE, Golbe LI, Lazzarini AM, Mark MH, Currie LJ, Wooten GF, et al. Epidemiologic study of 203 sibling pairs with Parkinson's disease: the GenePD study. *Neurology* 2002;58(1):79-84.
20. Uitti RJ, Calne DB. Pathogenesis of idiopathic parkinsonism. *Eur Neurol* 1993;33 Suppl 1:6-23.
21. Ben-Shlomo Y. The epidemiology of Parkinson's disease. *Baillieres Clin Neurol* 1997;6(1):55-68.
22. van der Kolk BA, McFarlane AC, Weisaeth L, editors. *Traumatic stress: the effects of overwhelming experience on mind, body, and society*. New York: Guilford, 1996.
23. McClain MT, Arbuckle MR, Heinlen LD, Dennis GJ, Roebuck J, Rubertone MV, et al. The prevalence, onset, and clinical significance of antiphospholipid antibodies prior to diagnosis of systemic lupus erythematosus. *Arthritis Rheum* 2004;50(4):1226-32.
24. Bonifacio E, Bingley PJ, Shattock M, Dean BM, Dunger D, Gale EAM, et al. Quantification of islet-cell antibodies and prediction of insulin-dependent diabetes. *Lancet* 1990;335:147-149.
25. Bennet PH, Rewers MJ, Knowler WC. Epidemiology of diabetes mellitus. In: Porte DJ, Sherwin RS, editors. *Ellenberg and Rifkin's diabetes mellitus*. 5th ed. Stamford, CT: Appleton & Lange, 1997:373-400.
26. Shenk D. *The forgetting. Alzheimer's: Portrait of an epidemic*. New York: Doubleday, 2001.
27. Duvoisin RC, Eldridge R, Williams A, Nutt J, Calne D. Twin study of Parkinson disease. *Neurology* 1981;31(1):77-80.
28. Ledoux J. *The emotional brain: the mysterious underpinnings of emotional life*. New York: Touchstone, 1996.
29. Levine P. *Waking the tiger*. Berkeley: North Atlantic Books, 1997.