Cross-Pathology Paradigms in Endocrinology

Gerald H Lushington*

C.S.O and E.V.P, TheraPeptics LLC, Lawrence, KS, USA

*Corresponding Author: Gerald H Lushington, C.S.O and E.V.P, TheraPeptics LLC, Lawrence, KS, USA.

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Most of us are aware that chemicals in our diet and environment can increase the risk of cancer, reproductive issues, developmental issues, and metabolic and immune disorders. What fewer people fully grasp is how many of these offending chemicals are endocrine disruptors - substances that alter hormonal balances in our bodies.

My own studies in neurology and immunology have peripheral overlap with endocrinology, yet I find the latter to be an interesting and immensely complex discipline. To put it in context, I like to compare the endocrine system to a country's federal civil service - an organization that neither makes the laws nor produces vital resources, but rather implements laws and controls how those resources are allocated.

Like the civil service, endocrine function covers a lot of responsibility, which means that when something disturbs our body, the endocrine system is affected. Thus, there are many distinct endocrine disorders, and many distinct endocrine disruptors contributing to these maladies. Endocrinology is so diverse, it almost seems like a catch-all umbrella for any pathology that isn’t explicitly listed within more targeted disciplines like oncology, neurology and epidemiology.

In truth, there are legitimate organizing principles in endocrinology, and numerous common markers connect distinct endocrine disorders. Indeed, it is the opinion of this editorial that seeking commonalities across distinct semi-related endocrine disorders may foster compelling new medicinal insight that might never come to light if we merely tried to separately catalog the entire etiology of each and every minor disease variant.

What brings me to this unusual claim? In fact, my statement is inspired by an eruption of biotechnology (see Becker’s list of 100+ artificial intelligence (AI) healthcare companies [1]) exploiting sophisticated new informatics techniques, for which broad analogy becomes more powerful than local certainty [2].

Biomedical research remains highly specialized, and is often quite ‘silied;’ but the promise of AI has rekindled interest in general trends. Where old machine learning methods sought clean classifications from focused data, powerful new deep learning (and related) algorithms mine heterogeneous information toward analogies across disparate phenomena.

To imagine what this might look like, consider an AI-assisted team uncovering novel treatment options for Graves’ ophthalmopathy (a rare disease, lacking extensive data) by mining diverse disease pathway characteristics for distantly related pathologies such as glaucoma, acromegaly and hyperthyroidism. Such cross-pathology learning embraces the broader focus of examining different diseases for similar symptoms and biomarkers that AI can then assess commonalities for relevance, thus potentially opening doors to unexpectedly applicable treatment paradigms.

Conceivably, this special journal edition of diverse topics in endocrine disruption could provide insight that, like wildfire leaping a barrier, will spark productive inquiry in a distinctly different subdiscipline. It is difficult to guess what illuminating spark may affect which application but, for the sake of stirring imagination, let me speak briefly about interesting problem on the cusp of neurology, immunology and endocrinology: the inflammation/glycation/adipogenesis cycle.

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This cycle is a key part of diabetes pathology. While diabetes is the single most prevalent endocrine disorder in the United States, it is better known by immunologists as an inflammatory metabolic disease. Specifically, the standard marker for diabetes is dysregulated glucose metabolism, which causes systemic profusion of glycated proteins [3], inciting oxidative stress and visceral adipocyte hypertrophy [3,4].

Or is it vice versa? It is equally valid to argue that pre-existing visceral adipocyte hypertrophy may promote insulin resistance, leading to profuse glycation and subsequent inflammation [5]. This ambiguity implies classic causal feedback, where downstream and upstream pathway effects intertwine, complicating the task of identifying a single root cause to target.

Yet, might endocrinologists help break this impasse?

Indeed, endocrine disruptor exposures are risk factors for both type 1 [6] and type 2 diabetes [7]. Admittedly, we lack strong consensus for precisely which disruptors pose the greatest diabetic risk, and which endocrine gland they perturb. Apparently, some disruptors affect adipose distributions, others directly trigger inflammation, while still others amplify systemic glycation.

Yet, even as etiology grows muddier, we recall that this is precisely the sort of conundrum that advanced AI methods are designed to resolve. Thus, unraveling the complex, multifactorial nature of diabetes might benefit from identifying analogies with simpler endocrine disorders, and comparing known pathway perturbations arising from endocrine disruptors.

And what might we learn from such comparisons?

Are there answers hidden somewhere in the following pages?

**Bibliography**