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No, You Can't Just Peek in Their Brain to See Their Future

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FYH 102: Blame it on the Brain

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It's thanks to adenine, guanine, cytosine and thymine that I have ten fingers, two eyes, and a uterus. They've got it coded neat and clear in my DNA double helix. It seems so straightforward, almost too simple, that the complexity of life could be captured by a twisting ladder of nucleotides. If this were true, genetic determinism argues that, because all events are casual and all personal information is described by genes, a person's future is decided by their genetic code. But human life moves fast and ends quick; evolution is too slow. It can't write the ancient DNA in each of our cells in a single generation. That's far too risky. Instead, our genes are regulated under a sort of accelerated evolution, epigenetics, which turns genes "on" or "off" in conversation with the environment. So, when people say that genetic determinism is reductionist, science has got a cutting edge counter argument in its back pocket: epigenetics. Epigenetics makes genes flexible, moderates their expression with the input from real life experiences (Waggoner & Uller, 2015, p. 2). Though, from a practical standpoint, can this new field really pick up the pieces left behind by genetic determinism? The "epi-" prefix seems like a cure-all, but how much could it really help to the end of predicting behavior? In order for behavior to be predicted accurately, all pertinent present variables need to be analyzed and disseminated into neat columns of data and fed into an algorithm. "Epi-," as defined by current science, is very broad and very vague (Waggoner & Uller, 2015, p. 11). It isn't made clear what exactly "environment" encompasses or how life experience could be enumerated and processed into future predicting machines. Epigenetic determinism may hold theoretically true, but does it have the potential to reap much practical reward? In order to answer this question, it's important to understand how predictive science gets its answers.

In behavioral psychology, predictive science is distinct from causal or explanatory research. In this field, casual research uses repeated correlation between a psychological event

and a faucet of genes, brain, or surroundings to try and draw out a relationship. Through many trials, it might be concluded that a reduced somatic response contributes to poor decision making in psychopaths or so on. However, this sort of data performs very poorly when used to build predictive models. Explanation based-models drawn from meticulous research are worse at prediction than crudely constructed, demographic models (Yarkoni & Westfall, 2017, p. 1).While much of psychological research examines the causes of behaviors in order to find an explanation for them, predictive research creates models able to predict behavior with varying amounts of accuracy (Plomin & von Strumm, 2022, p. 1). Causal or explanation-based research does not have predictive power. Predictive research does not provide an explanation or a causal link (Plomin & von Strumm, 2022, p. 2). This means that predictive models don't provide the "why," and causal research can't predict the "when and where." In fact, predictive research relies so little on the "why" that the AI algorithms which produce these models are totally opaque. Researchers can't explain the gap between initial data and predicted outcome.

In combination with this mystery, predictive models are also often flawed. They work best when developed on a macro scale (Plomin & von Strumm, 2022, p. 3). This research wasn't made for the individual. Complex traits are especially difficult to predict because their heritability sits at around 50% (Plomin & von Strumm, 2022, p. 2). That means it makes it very hard for accurate results to be produced from plugging in genetic or epigenetic data. Furthermore, the larger scale a study is, the more likely it is to have a weak behavioral connection because reducing overfitting means eliminating trends (Yarkoni & Westfall, 2017, p. 9). A binge drinking study with a sample size of 692 predicted drinking in young adults with only 70% accuracy, despite its 2 year data collection in behavior, genetics, and neuroimaging (Yarkoni & Westfall, 2017, p. 9). While 70% might seem to be significant, the same level of

accuracy could be reached simply by asking if the subject already smoked (Yarkoni & Westfall, 2017, p. 10). Plugging someone into these models provides–at best–a crude idea of future behavior. At worst, current science produces a result punctured by biases. Using these predictive models on an individual basis is very inaccurate: so why are courts pretending like it makes sense?

Imagine this: you're a prosecutor in a crazy-cool high profile case. All you need is rock-solid evidence to put your suspect behind bars. Cognitive neuroscience seems to provide a straight-shot sort of answer: just look into their brain. But that's the huge problem with the far-flung promises of predictive science and neuroimaging. Brain scans look so concrete and inarguable, yet they are riddled with the same small-scale inaccuracy and biases. Unrealistically, up to 90% of published neuroimaging research has statistical significance (Javala et al., 2023, p. 2). This high experimental "success" rate is in part due to pressure from journals, which prefer more groundbreaking results (Javala et al., 2023, p. 2). Human researchers are vulnerable to manipulating data, in this case through procedural overfitting (Yarkoni & Westfall, 2017, p. 5). 46% of psychologists surveyed confessed to selectively reporting successful studies, and 38% confessed to excluding data after analyzing the results (Yarkoni and Westfall, 2017, p. 5). Modeling a researcher's bias found that procedural overfitting can introduce a rate of 50% or more false positives (Szucs & Ioannidis, 2023, p. 14).

However, this bias could also be due to a smaller sample size. Cognitive neuroscience experiments are usually confined to smaller numbers because of the increased costs and tedious data collection. The data collected can undergo more complex and flexible analysis than that of a psychology or medical journal's studies (Szucs & Ioannidis, 2021, p. 13). These smaller sample sizes make overfitted data, inventing trends, and creating a model that, while accurate to a

specific experimental trial, cannot be used to predict future results. (Yarkoni & Westfall, 2017, p. 3). For example, one study of alcoholism found alcoholics experience stimulus in the bilateral putamen, bilateral ACC, adjacent medial prefrontal cortices, and bilateral visual secondary cortices with an intensity that nonalcoholics do not (Grüsser et al., 2004, p. 299). On the surface, this seems like compelling evidence for a dramatic structural change in the brains of alcoholic adults. However, this study was conducted with a sample size of only 10 alcoholics, making it a compelling call for future research, but allowing too much room for overfitting to provide accurately predictive results (Grüsser et al., 2004, p. 297). Evidence like this couldn't be used to make the case that a single alcoholic's brain is irreparable damaged by their alcohol consumption, for example, especially in the face of epigenetic and neuroplasitic opportunities during sobriety. For the same reason, it would be unethical to pin guilt on a criminal based on how they perform in brain scans.

Javala et. al recently evaluated 64 relevant psychopathy studies, only one set of which satisfied these conditions: large scale (more than 20 participants per group or 80 participants in a single group) and reproducibility (the presence of at least 2 studies which use a large enough scale and are relevant to the subject (2023, pp. 5-6). This has big implications for the believed validity of brain imaging, as it is often presented as "hard evidence," despite the common biases (Szucs & Ioannidis, 2021, p. 13). Reproducibility restrictions on evidence in court would ensure that brain imaging studies referenced have an appropriate amount of follow-up studies. Doing so might encourage researchers to target bias in their research; journals may re-evaluate the low priority placed on replication (Javal et al., 2023, p. 12). There are similar limitations present in neuroimaging-based clinical depression research. Apart from low power studies, neuroimaging-based clinical depression research is also hampered by its reduced scope. By

integrating clinical and genetic data, a much more accurate diagnosis can be determined (Kang & Cho, 2020, p. 8). Neuroimaging, though it may seem to tell the whole story, is not powerful enough to actually do so. Brain imaging evidence is–as a whole–a newly developing field and many published studies lack high enough powers or recorded reproductions to gain enough predictive integrity. Biases occur both procedurally and statistically, making it impossible to use brain evidence as a concrete cure-all.

This means that you–the ambitious prosecutor that you are–shouldn't be allowed to stand up and argue that a brain scan proves your suspect guilty. Scientific evidence in court is evaluated through the Daubert Standard, though the Frye Standard is still used in some state courts (Robinson, 2023, para. 3). The Frye Standard requires evidence to be generally accepted by many experts in the field, but the Daubert Standard requires proven replicability, widespread professional acceptance, experimental standards, potential error rate, publication, and peer review (Robinson, 2023, para. 4). On the face of it, the described neuroimaging and behavioral genetics studies do not meet this standard; they lack proven replicability. However, because of the vague standards set for replicability and potential error rate, science that should not have been referenced has been used in court. In the case State v. Brian Dugan, the defense originally presented fMRI data to combat the death penalty. Rather than refuting its use, the prosecution used the evidence to further incriminate Dugan; a jury sentenced him to death–though this was later repealed by the state (Javala et. al, 2023, p. 4). Knowing the flaws present in neuroimaging research, holding Dugan or Gare Bear at greater responsibility based on their brain scans risks stigma.

Predictive research, explanatory research, and neuroimaging hold potential for alleviating criminal accountability. However, they can not have a place in establishing guilt. To do so also

invites undue social stigma onto the suspect as well as others with mental illness. Where science is unable to provide a definite yes or no, behavioral genetics has a history of infecting the gaps with society. Classical eugenics manipulated behavioral genetics to fit the white supremacist pseudo-scientific concept of race (Liscum & Garcia, 2022, p. 905) Mental illnesses like substance use disorder to psychopathy contribute to the crimes committed, but they do not make that behavior inevitable. Neuroplasticity means that brain scans aren't static; the complexity of an individual cannot be captured by a predictive model. Society leaks into science, and having loose regulations on reproducibility or treating predictive evidence like certainty discourages recovery or treatment by producing social stigma.

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