APEIRON
CENTER FOR HUMAN POTENTIAL

Sleep Genotype Report

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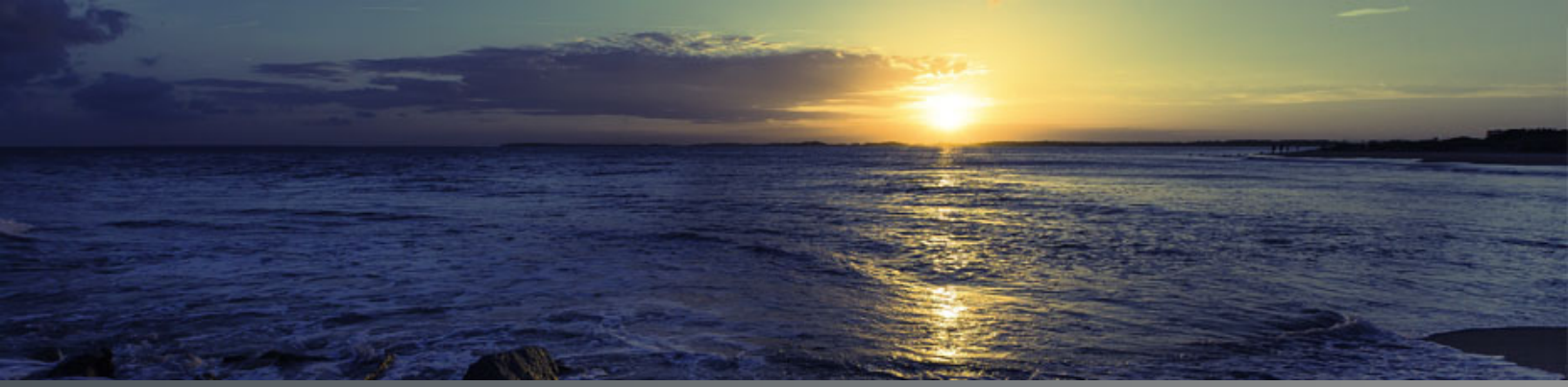


CIRCADIAN PROPENSITY

GENETIC DATA

GENE	GENO TYPE
PER2(1)	CG
PER2(2)	CC
PER3(1)	AA
PER3(2)	CC
AANAT	GG

MORNING

YOUR CHRONOTYPE

Humans are a diurnal species. Generally, we are active during the day and sleep at night. Many us feel more awake, alert and capable of our best work effort in the morning. However, there are those at the opposite end of the spectrum who prefer to stay up late and sleep well into the daytime hours. These indiv-i duals find themselves most alert in the evening.

The circadian rhythm is a cycle that signals our bodies when to sleep, rise and eat. Individual circadian variations are governed by the circadian rhythm through the internal circadian clock network. This internal biological clock network resides in the brain and regulates the timing of functions such as appetite, hormone release, and metabolism. Our perception of this clock is the basic sleep-wake process.

Recent research has revealed that the circadian clock is not as basic as we once suspected. While we have tendencies toward a basic rhythm, what determines our desire to wake early or stay up late, is influenced by the same system that regulates the cycling of many bodily functions. Forcing the body to fit into a sleep-wake cycle that does not match our genetics, can lead to circadian dyssynchronization

INTERPRETATIVE NOTES:

Definite morning propensity, meaning that your cognitive function and likely your physical performance will be better in the early parts of the day.

SLEEP ONSET

GENETIC DATA

GENE	GENO TYPE
NPSR1	AT
CLOCK(3)	AG
PER3(1)	AA
PER3(2)	CC
AANAT	GG
COMT	GG

TYPICAL PROPENSITY

Normal sleep latency, the time from lying down to the first stages of sleep, is approximately 15-20 minutes. Falling asleep faster indicates a degree of sleep deprivation. Often, individuals with a genetic propensity for later sleep, try to force themselves into a different chronotype (circadian rhythm) which can result in significant sleep onset delays

Individual genetics play a large role in the prediction of longer or shorter periods of sleep onset. Despite possessing a propensity for delayed sleep onset, identifying the genetic components that are most impactful to the process allows a much more directed and personalized approach to optimal sleep interventions.

Normal time to onset of sleep is about 15-20 minutes. Onset of sleep outside of this range usually indicates either a genetic or lifestyle component

- 0-5 minutes = severe sleep deprivation
- 6-15 minutes = moderate sleep deprivation
- 15-20 minutes = normal
- >20 minutes = probable genetic or environmental component

INTERPRETATIVE NOTES:

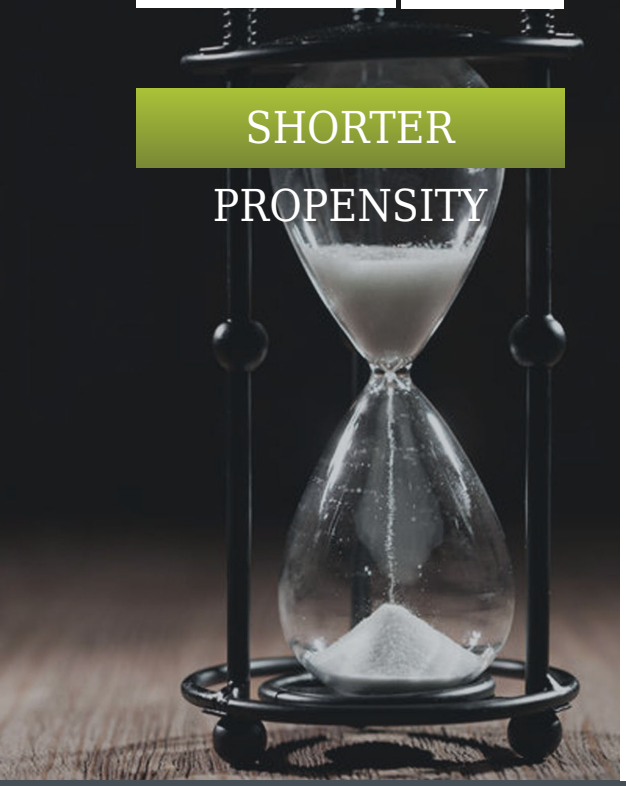
Normal propensity for a 10-20 minute onset of sleep, shorter or longer time frames should prompt an investigation into lifestyle factors.

SLEEP DURATION

GENETIC DATA

GENE	GENO TYPE
NPSR1	AT
CLOCK(1)	GG
CLOCK(2)	AG
PER2(1)	CG
GNB3	CT
ADA	CC
ABCC9	TT
GRIA3(1)	CC
COMT	GG

SHORTER PROPENSITY



When we don't get the sleep we need, we experience surges of stress hormones which disrupt our cognition and ability to regulate emotions. 90% of adults require 7 - 9 hours of sleep a night however, most do not achieve this mark.. Lost sleep reduces brain power and productivity, diminishes concentration and impairs memory. It lowers creativity, reduces the ability to communicate, impairs motor skills and increases stress and anxiety

Studies have demonstrated that just two hours of sleep deprivation (5 - 6 hours of sleep) results in a vigilance level equivalent to the consumption of two alcoholic drinks. Interestingly, while there is a detrimental decline in vigilance our perceived level of vigilance will be normal.

A multitude of factors contribute to how long we sleep. Assessing your genetic sleep variations and establishing effective sleep strategies are essential steps in the process of sleep optimization. When you know your genetic propensities for sleep it becomes easy to create a strategic sleep plan and achieve beneficial sleep.

Sleep requirements:
Teens 9-10 hours
Propensity Adults 7-9 hours

INTERPRETATIVE NOTES:

Slightly less need for sleep so getting 7-8 hours of total asleep time should work well.

QUALITY OF SLEEP

GENETIC DATA

GENE	GENO TYPE
TNFa	GG
BDNF	CC
MTNRB	CC
PPP2R4	GG
ADORA2A	CT
GRIA3(1)	CC
COMT	GG

LOW

PROPENSITY FOR DISRUPTED SLEEP

Sleep Matters. It is the single most important thing you can do to improve performance in life. Sleep quality is determined by a complex network of interacting physiological processes which are strongly influenced by lifestyle. The quality of our sleep is influenced by the amount of deep sleep, the number and duration of waking episodes and the number of REM episodes. When our lifestyle is not in sync with our chronotype, sleep quality can be significantly impacted.

Genetic predispositions can evaluate variables such as melatonin production and response, excitatory versus inhibitory neurotransmitters and responses to day environment - light, caffeine, etc. Once a genetic variable is identified, a directed approach can be taken to improve the underlying issue. It is important to first get sleep patterns matching before addressing sleep quality.

Lifestyle modifications and precision nutrigenomic interventions are highly successful for addressing genetic variations that contribute to sleep quality. Improvements in sleep quality translate to improvements in all aspects of performance

HIGHLIGHTS

Low risk for disrupted sleep due to genetics.

NARCOLEPSY RISK

GENETIC DATA

GENE	GENO TYPE
HLA-DRB1 TRCA	GG TT

TYPICAL RISK

Narcolepsy is a long-term neurological disorder that involves a decreased ability to regulate sleep-wake cycles. It has been estimated that 1 in 2000 people are affected by narcolepsy. There are two types of narcolepsy:

Type 1

Irrepressible need to sleep or daytime lapses into sleep occurring for ≥3 months.

The presence of one or both of the following

- Cataplexy (paralysis of motor control) and a mean sleep onset of ≤8 minutes and ≥2 sleep-onset REM periods.
- Spinal fluid hypocretin-1* concentration is either ≤110 pg/mL.

Type 2

- Daily periods of irrepressible need to sleep occurring for ≥3 months
- A mean sleep latency of ≤8 minutes and ≥2 sleep-onset REM periods.
- Cataplexy is absent
- CSF hypocretin-1 concentration has not been measured or concentration >110 pg/mL

*Orexin, also called hypocretin, is a neuropeptide that regulates arousal, wakefulness, and appetite.

INTERPRETATIVE NOTES:

No increased risk

RESTLESS LEG

GENETIC DATA

GENE	GENO TYPE
MEIS1	GT
BTBD9(1)	CT
BTBD9(2)	GG
MAP2K5(1)	GG
MAP2K5(2)	GG
PTPRD	AG

TYPICAL

PROBABILITY

Restless leg syndrome (RLS) is a disorder that causes a strong urge to move one's legs. It is characterized by an overwhelming need to move the affected limb and symptoms tend to be worse at night. Sensory symptoms are triggered by rest, relaxation, or sleep and are relieved with movement which persists if movement continues

Potential causes of RLS include iron deficiency, genetic predisposition, brain neurotransmitter imbalances and increased brain glutamate. There are no specific tests to confirm RLS and a medical professional is required to assess and diagnose its presence. The above criteria are only suggested guidelines to seek medical confirmation.

Carrying genetic predispositions towards restless leg syndrome does not suggest that it is present, this is solely based on probability. There will be many people that have the symptoms of RLS without a genetic predisposition as well. The genetic variations can potentially guide your clinician or coach in designing interventions that will have a higher probability of mitigating the symptoms.

HIGHLIGHTS

Normal risk for limb movements during sleep.

RECOMMENDATIONS