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# Running the Cape Town Procedure for 2020

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#### **Background** 1

This paper details the key data inputs to the Cape Town Procedure (CTP) [1], the TAC calculation given the agreed data, and the breakdown of the MP calculation.

#### 2 **Data inputs**

There are four data inputs in the CTP:

- 1. Gene tagging: the MP uses the abundance estimate and the number of matches associated with that estimate
- 2. Japanese LL CPUE: the agreed Japanese long-line CPUE series
- 3. CKMR POPs: the updated parent-offspring pairs
- 4. **CKMR HSPs**: the updated half-sibling pairs

#### 2.1 Gene tagging

We now have three years of gene tagging 2 year old abundance estimates for the years 2016, 2017, and 2018. Table 2.1 details the estimates and the number of recaptures associated with the estimate as these are the inputs to the MP.

Year	Estimate	Recaptures		
	2.27e+6	20		
	1.15e+6	67		
2018	1.14e+6	66		

Table 2.1: Gene tagging abundance estimates and associated recaptures, both used as inputs to the MP.

### Japanese long-line CPUE

The CPUE used in the CTP is a simple arithmetic mean of the w0.5 and w0.8 individual CPUE series. Figure 2.1 shows the CPUE index used as an input to the CTP.

#### **CKMR POPs & HSPs** 2.3

A more detailed summary of the two CKMR data sets, as well as the fit of the adult population dynamics in the CTP to these data, can be found in [2]. For the POP data set we know have a juvenile cohort coverage of 2002-2015 (i.e. these are the years for which we have informative data on the spawning stock), with around 112 million comparisons and 89 detected POPs. For the HSPs we have data covering the juvenile cohorts of 2003–2014, there are around 88 million juvenile comparisons and 115 cross-cohort HSPs. The false positive threshold used to rule out lesser kin such as half-cousins (HCPs) in the HSPs has been increased, given the additional comparisons now being undertaken. The subsequent false negative retention probability (i.e. fraction of true number of HSPs in the data set above the false positive threshold) has been decreased from 0.84 previously to 0.74. So the actual number of HSPs in the data for 2020 is essentially the same as 2019, but the true number of HSPs has increased in such a manner as to be consistent with the previous data set used in 2019. That is not to say we haven't increased the

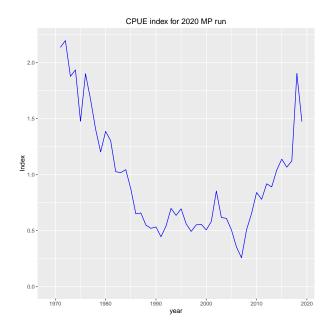


Figure 2.1: CPUE index used as input to the 2020 MP.

level of information in the HSP data - we have an additional year of adult abundance information and better information on mean adult Z given the increase in longer comparison timeframes when it comes to the juvenile birth years in the data. The hit rates (matches-per-comparison) for both the POPs and HSPs are slightly lower than the 2019 data, which is qualitatively suggestive of a slight overall increase in the adult population.

## 3 Structure of the Cape Town Procedure

The general structure of the CTP is as follows:

- The MP uses CPUE, gene tagging and CKMR (POP and HSP) data
- For the CKMR part a simplified adult population model (abundance and total mortality) is fitted to the CKMR data. The log-linear trend in TRO,  $\lambda^{\rm ck}$ , is then used in the HCR. Prior to the estimated recovery of the stock to the tuning a level TAC increases are permitted *only* for positive trends above a minimum positive level. As the stock reaches the target level this reverts to positive/negative trends increasing/decreasing the TAC.
- For both the CPUE and CKMR trend terms the gain parameter is density-dependent. For a given level of TRO rebuilding (relative to the recent estimates) the gain parameter is stronger prior to reaching the rebuilding then decreases as the TRO reaches the target level. This ensures reactivity when needed (in the rebuilding phase) but stability when rebuilding is achieved.
- For the gene tagging term a limit-type approach is used: (i) for values of the current 5 year average 2-year old abundance below the limit strong (supralinear) decreases in TAC are enacted; (ii) for values above the upper level weaker (sublinear) increases in TAC are permitted; (iii) for values between the two nothing is done to the TAC. A crucial difference for the GT part of the HCR is that there is no inertia: once the values appear outside the bounds of inaction the TAC is proportionally changed

 For the CPUE data the HCR is similar to that applied to the gene tagging data: there is a zone where no change is recommended and above and below this level of mean recent CPUE there is a linearly increasing or decreasing change in TAC

The general structure of the revised MP is as follows:

$$TAC_{y+1} = TAC_y \left( 1 + \Delta_y^{\text{cpue}} + \Delta_y^{\text{ck}} \right) \times \Delta_y^{\text{gt}}, \tag{3.1}$$

where the inertial terms for the CPUE and CKMR parts of the HCR are additive, not multiplicative. This avoids the quadratic term in the multiplicative case where both trends are consistently positive consistently making the TAC increases larger than for the additive case, despite the trends being the same in both cases. Before detailing the functional form of the HCR we recap some useful variables:

- ullet  $I_y^{
  m ck}$ : moving average (of length  $au^{
  m ck}$ ) of the estimated TRO from the MP population model (projected forward to the current year using the model to project forward for 4 years to avoid too much intertia in the signal when you need it)
- I: average estimated TRO from 2003 to 2014 (reference period w.r.t. relative rebuilding criterion)
- $\gamma$ : proportional amount of TRO rebuilding we wish to achieve
- $\eta = I_y^{\rm ck}/(\gamma \tilde{I}) 1$ : the variable at which passing from negative to positive indicates the point at which the TRO rebuilding has been achieved and the transition in the reactivity of the MP occurs (i.e. it goes from reactive to passive w.r.t. CPUE and CKMR signals only)

For the CPUE part of the HCR we used a density-dependent gain parameter:

$$k^{\text{cpue}}(\eta) = w_1^{\text{cpue}} \left( 1 - \left( 1 + e^{-2\kappa\eta} \right)^{-1} \right) + w_2^{\text{cpue}} \left( 1 + e^{-2\kappa\eta} \right)^{-1}$$

This is using the logistic function approximation to the Heaviside step function  $H[\eta]$  ( $H[\eta < 0] =$  $[0, H[\eta \geq 0] = 1)$ . We set  $\kappa = 20$  so the transition between the two gain parameters, given  $\eta$ , happens within  $\pm 5\%$  of  $\delta = 1$ . The CPUE multiplier is then just defined as follows:

$$\Delta_y^{\text{cpue}} = k^{\text{cpue}}(\eta) \left( \delta_y^{\text{cpue}} - 1 \right)$$

and  $\delta_y^{
m cpue}$  is actually very similar in form to the gene tagging part of the HCR

$$\begin{split} \delta_y^{\text{cpue}} &= \left(\frac{\bar{I}_{\text{cpue}}}{I_{\text{low}}}\right)^{\alpha_1} \quad \forall \bar{I}_{\text{cpue}} \leq I_{\text{low}}, \\ \delta_y^{\text{cpue}} &= 1 \qquad \quad \forall \bar{I}_{\text{cpue}} \in \left(I_{\text{low}}, I_{\text{high}}\right), \\ \delta_y^{\text{cpue}} &= \left(\frac{\bar{I}_{\text{cpue}}}{I_{\text{low}}}\right)^{\beta_1} \quad \forall \bar{I}_{\text{cpue}} \geq I_{\text{high}}, \end{split}$$

where  $ar{I}_{
m cpue}$  is the (4 year) moving average LL1 CPUE,  $ar{I}_{
m low}$  and  $ar{I}_{
m high}$  are upper and lower threshold CPUE values, and  $\alpha_1$  and  $\beta_1$  allow for an asymmetric response above or below the threshold zone.

For the CKMR part of the HCR we try to ensure a minimum rate of increase in the TRO beneath the target level, and once it is achieved we would like to maintain the TRO at that level. To

include this kind of behaviour in the HCR we also include some density-dependence in the loglinear growth rate at which the HCR moves from a TAC increase to a TAC decrease:

$$\begin{split} & \Delta_y^{\text{ck}} = k^{\text{ck}}(\eta) \left( \lambda^{\text{ck}} - \tilde{\lambda}(\eta) \right), \\ & k^{\text{ck}}(\eta) = k_1^{\text{ck}} \left( 1 - \left( 1 + e^{-2\kappa\eta} \right)^{-1} \right) + k_2^{\text{ck}} \left( 1 + e^{-2\kappa\eta} \right)^{-1}, \\ & \tilde{\lambda}(\eta) = \lambda_{\min} \left( 1 - \left( 1 + e^{-2\kappa\eta} \right)^{-1} \right) \end{split}$$

The threshold level at which the log-linear trend,  $\lambda^{\rm ck}$ , goes from supporting a TAC decrease to an increase essentially begins at  $\lambda_{\rm min}>0$  and, as the estimated TRO approaches the target level, rapidly decreases to zero (in a similar way to the CPUE trend term). This is to ensure that a minimum level of rebuilding is encouraged for **all** trajectories below the target, and where above the target the *status quo* is preferred.

To calculate the recent mean age 2 abundance from the gene tagging data consider a weighted moving average approach:

$$\bar{N}_{y,2} = \sum_{i=y-1-\tau^{\text{gt}}}^{y-2} \omega_i \widehat{N}_{i,2}$$

where  $\omega_i$  is a weighting proportional to the number of matches used to produce the GT estimate  $\widehat{N}_{i,2}$  (basically inverse variance weighting). The 2 year delay between having the estimate and what year it actually refers to is factored into the calculation. The multiplier for the GT part of the HCR is as follows:

$$\begin{split} & \Delta_y^{\text{gt}} = \left(\frac{\bar{N}_{y,2}}{N_{\text{low}}}\right)^{\alpha} & \text{if} \quad \bar{N}_{y,2} \leq N_{\text{low}}, \\ & \Delta_y^{\text{gt}} = 1 & \text{if} \quad \bar{N}_{y,2} \in (N_{\text{low}}, N_{\text{high}}), \\ & \Delta_y^{\text{gt}} = \left(\frac{\bar{N}_{y,2}}{N_{\text{high}}}\right)^{\beta} & \text{if} \quad \bar{N}_{y,2} \geq N_{\text{high}} \end{split}$$

with  $N_{\rm low}$  the limit level and  $N_{\rm high}$  the upper level at where TAC increases are permitted. Table 3.1 details the parameter values for the HCR in the adopted MP.

### 4 TAC calculation and breakdown

The Cape Town Procedure standalone ADMB code was run with the agreed 4 data inputs (gene tagging, Japanese LL CPUE, CKMR POPs and HSPs). For the given initial parameter guess the maximum likelihood estimate of the adult population model fitted to the CKMR data was attained (maximum gradient 4.72e-5) and showed essentially no sensitivity to close-by alternative initial parameter guesses. Given the conclusion that the CKMR data were more than adequately explained by the adult model [2], we would suggest that there are no complications with the model-based part of the CTP. Table 4.1 details the influence breakdown (in terms of a TAC multiplier purely for that part of the HCR) for each of the three components of the CTP, as well as the current and suggested TAC when all components are combined together as per Eq. (3.1).

Parameter	Value		
$ au^{ m cpue}$	4		
$w_1^{ m cpue}$	0.9		
$w_2^{ m cpue}$	0.005		
$I_{ m low}$	0.45		
$I_{ m high}$	1.42		
$\alpha_1$	1		
$\beta_1$	1		
$ au^{ ext{gt}}$	5		
$N_{ m low}$	1e+6		
$N_{ m high}$	2.6e+6		
$\alpha$	1.5		
$\beta$	0.25		
$ au^{ m ck}$	3		
$ au^{ m ck} \ k_1^{ m ck}$	1.25		
$k_2^{ m ck}$	0.05		
$\gamma$	1.5		
$\lambda_{\min}$	0.001		
$\kappa$	20		

Table 3.1: Fixed values of parameters of the HCR in the CTP.

Variable	Value
$\Delta^{ m gt}$	1
$\Delta^{ m cpue}$	1
$\Delta^{ m ck}$	-6.6e-4
$(1 + \Delta^{\text{cpue}} + \Delta^{\text{ck}}) \times \Delta^{\text{gt}}$	0.99934
Current TAC	17,647t
Suggested TAC	17,647t

Table 4.1: Breakdown by HCR component, and the current and suggested TACs.

For the gene tagging, the (3 year) recapture-weighhed average age 2 abundance is 1.29 million, so within the 1-2.6 million region where the TAC multiplier is 1. For the CKMR the log-linear trend is just below the minimum required level (but still positive) and suggests a very small reduction in TAC. For the CPUE index, the 4 year mean (2016-2019) is 1.39, and the threshold value at which the CPUE part of the HCR wants to increase the TAC is 1.42; as a result, this part of the HCR recommends no TAC change. The combined suggested change in TAC is -11.6t which is well below the 100t minimum change so the implied TAC will be 17,647t i.e. no change.

#### **Discussion** 5

This paper detailed the structre of the Cape Town Procedure, the four data sets used as inputs (gene tagging, Japanese long-line CPUE, CKMR POP and HSP data), and how they all link together within the adopted MP to give the suggested TAC for the 2021-2023 period.

## 6 Acknowledgements

This work was funded by CSIRO and the Australian Fisheries Management Authority.

## **References**

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